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Research Paper

Respiratory Failure in Critically Ill Pregnant Women

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ABSTRACT
As many as 0.2 percent of pregnancies are affected by respiratory failure, with the majority occurring after delivery. The examination and treatment of these individuals are impacted by the mother's altered respiratory physiology. As well as harming the mother, acute respiratory failure during pregnancy can also harm the fetus because it interferes with the fetus's ability to receive oxygen. Women who become critically ill while pregnant face unique issues that must be addressed. When a woman is pregnant, her body goes through a variety of changes that might damage her cardiovascular and pulmonary systems. Changes might lead to life-threatening complications when they interact with preexisting or new comorbidities. Considering the effects of disease and therapy on fetal growth and outcome must also be considered constantly. During pregnancy, disorders include peripartum cardiomyopathy, amniotic fluid embolism, preterm labor, oncolytic therapy-induced acute pulmonary edema, intraamniotic infection, and ovarian hyperstimulation syndrome can cause respiratory failure.
KEYWORDS:*Pregnancy, obstetric, respiratory failure, ventilation, fetus, embolism, critical illness, eclampsia, pulmonaryedema.*

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I. INTRODUCTION

Pregnancy causes a slew of changes to the body's structure and function, including the respiratory system. These alterations frequently have an impact on how various respiratory infections present and are treated during pregnant. There is a decrease in carbon dioxide levels during pregnancy compared to the non-pregnant condition, and the diaphragm rises by 4-5cm [1].

Lung volume changes dramatically during pregnancy. tidal volume (TV) increases by 30 to 50 percent, albeit at the cost of functional residual capacity (FRC) During pregnancy, minute ventilation (the product of respiratory rate and TV) is reduced from 35-40 mmHg in the nonpregnant state to 27-32 mmHg, resulting in higher PaO2 and lower PaCO2 in the mother's circulation (104-108 mmHg) [2,3]. Because of this decrease in PaCO2, an increase in bicarbonate excretion by the kidneys compensates for the drop in serum bicarbonate. Lower bicarbonate levels move the hemoglobin oxygen dissociation curve to the right, which reduces the affinity of maternal hemoglobin to oxygen, making oxygen transport to the fetus easier [4].

Distinguishing between underlying disease and typical pregnancy-related dyspnea can be a tough diagnostic problem when a pregnant woman complains of dyspnea. Some causes of respiratory failure are unique to pregnancy as peripartum cardiomyopathy, amniotic fluid embolism, severe preeclampsia and ovarian hyperstimulation syndrome {OHSS}, tocolytic therapy induced acute pulmonary edema, chorioamnionionitis, endometritis [5].

Other conditions that can be affected by pregnancy and lead to dyspnea include: acute pulmonary edema, aspiration of gastric content, bronchial asthma and venous thromboembolism as pregnancy is hypercoagulable state, fungal infection also Bacterial andviral pneumonia [6].

Many other conditions unaffected by pregnancy can also cause respiratory failure during pregnancy as, ARDS, fat embolism, inhalational injury, sepsis, burns, acute pancreatitis and transfusion related acute lung injury (TRALI) [7].

II. OVERVIEW OF THE STUDY AND DISCUSSION

Per partum Cardio Myopathy (PPCM)

Heart failure symptoms appear in the final month of pregnancy and persist through the fifth month after delivery in the unusual case of per partum cardiomyopathy (PPCM). Heart failure must have no other known

cause, and there must be no known heart illness before the last month of pregnancy. In order to do an echocardiogram, one of the following conditions must be met: left ventricular end diastolic size >2.7 cm/m2 body surface area, with a probable additional ejection fraction 45 percent, or fractional shortening 30 percent, or both [8].

Since other etiologies have been recognized as having the potential to lead to PPCM, this diagnosis is now a more inclusive umbrella term that incorporates heart failure caused by a variety of illnesses that occur during the defined time span. Unbalanced oxidative stress and reduced angiogenesis appear to be a common element among the etiologies that have been found. Oxidative stress that leads to PPCM can be caused by high blood pressure, aberrant hemodynamic stress responses, viral etiology, and nutritional deficits [9].Normal pregnancy's symptoms might match those of mild heart failure, making it easy to miss the onset of PPCM. Distress, weariness, and peripheral edema are common in these individuals. Left ventricular failure leads to dilatation of the left ventricle, which leads to arrhythmias and embolic events [10]. Hepatomegaly, S 3 and S 4 gallop, and jugular venous distension are all symptoms of heart failure hypoxia in these patients [11].

PPCM-induced acute heart failure has yet to be studied in humans in clinical trials to determine how it should be treated. As a result, typical acute systolic heart failure therapy is used in these patients. Protecting the airway, managing breathing, and maintaining circulation are all critical in women with severe acute decompensated heart failure. When a patient has acute pulmonary edema, intubation or bilevel positive airway pressure may be required to give adequate oxygenation. Anti- and post-natal loop diuretics are both safe and effective at providing the necessary diuresis [12]. Afterload and preload can be reduced with continuous infusions of vasodilators like as nitroglycerin and nitroprusside. The use of an inotrope such as milrinone or dobutamine, or additional mechanical support (i. eintra-aortic balloon pump, left ventricular assist device, or extracorporeal membrane oxygenation) may be required for more severe cardiogenic shock if there is significant cardiac dysfunction [13].

Amniotic Fluid Embolism

It is estimated that 7.7 out of every 100,000 pregnancies result in an amniotic fluid embolism (AE). Severe dyspnea and hypoxia, followed by seizures and circulatory collapse or stoppage, are the most common symptoms of this condition, which most typically happens during or after childbirth or following uterine manipulation. ARDS and disseminated intravascular coagulation can occur in those who survive the first episode [14].

Meconium staining of amniotic fluid increases the risk of amniotic fluid embolism in older mothers, high parity, cesarean section, and low uterine segment laceration. A traumatic opening of uterine vessels may be the mechanism of amniotic fluid embolism, as revealed by data from a US registry of cases, where 78% of the patients had ruptured membranes and some had just undergone intrauterine treatments. However, this finding does not rule out alternative diagnoses as patients with no symptoms may also have fetal squamous cells recovered from pulmonary artery catheters at autopsy [15]. Pulmonary hypertension and left ventricular failure occur together hemodynamically, explaining the cause. Amniotic fluid elements such as leukotrienes and arachidonic acid metabolites may be to blame for these side effects. Anaphylactic syndrome of pregnancy has been proposed as a new name due to certain similarities to anaphylaxis. Low C1 esterase inhibitor levels may play a pathogenic and predictive function, according to new research [16].

Patients typically acquire bilateral lung infiltrates on x-rays. Amniotic fluid embolism does not have a specific treatment and is managed by providing supportive care for the concomitant disseminated intravascular coagulation and left ventricular and respiratory failure [17]. If the unborn child makes it through the early ordeal, it should be delivered as soon as possible. Emergency postmortem or peri resuscitative cesarean sections should be performed like in other cases of cardiac resuscitation during pregnancy if the mother is in imminent danger of dying from her condition. Maternal mortality rates have ranged from as high as 86% to as low as 22%, depending on the source. Maternal deaths caused by amniotic fluid embolism are thought to account for 14% of all maternal deaths [18].

Tocolytic Therapy-Induced Acute Pulmonary Edema

Tocolytic therapy-induced pulmonary edema usually happens when the medicine is taken orally or intravenously, or within 24 hours of stopping the beta-agonist medication. When tocolytic medication fails to stop labor, it's possible that respiratory problems will arise during the first 12 hours after delivery. Multiple pregnancies or concomitant conditions such as localized or systemic infections, silent heart disease, or treatment with magnesium sulfate or corticosteroid increase the incidence of this problem [19]. Chest pain and/or dyspnea are the most common symptoms, but so are a cough and pink or frothy sputum. Bilateral crackles are seen on the chest exam, and a heart exam usually reveals nothing. A chest radiograph reveals pulmonary edema-related bilateral alveolar infiltrates [20]. Fluid infusions used to control pharmacological therapy with beta-agonists'

systemic vasodilation may cause a volume overload if vasoconstriction develops after the therapy is stopped. Pathogenic pathways may be aided by the pulmonary capillary leak phenomena [21].

Postpartum pulmonary edema in cocaine addicts treated with bromocriptine to inhibit lactation should also take into account a newly reported entity of pulmonary edema. It is possible that oncolytic-related pulmonary edema is caused by the absence of clotting problems and the quick resolution of pulmonary edema [22]. Patient management should begin with oxygen supplementation and attentive observation, followed by intubation and ventilator assistance if the patient's condition worsens significantly. Patients with volume overload may benefit from diuretic therapy, whereas patients with intravascular volume depletion may experience hypotension as a side effect [23].

Preeclampsia

There are two types of preeclampsia: mild and severe. Mild cases occur before 20 weeks of pregnancy, whereas severe cases can appear as late as 4-6 weeks after delivery. Hypertension and proteinuria, with or without pathologic edema, are the main clinical symptoms [24]. Although there is no medical consensus on the parameters that characterize preeclampsia, appropriate criteria include an SBP larger than 140 mm Hg and a DBP greater than 90 mm Hg on 2 successive readings, 4-6 hours apart, in a woman who was normotensive before 20 weeks' gestation. Essential hypertension is identified as preeclampsia when the systolic blood pressure or diastolic blood pressure increases by 30 mm Hg or 15 mm Hg [25].

Pregnant women are more likely to develop pulmonary edema due to pregnancy's decreased serum oncotic pressure. Reduced serum oncotic pressure is a result of the 50% increase in blood volume during pregnancy, which is mostly achieved through an increase in plasma free water [26]. As a result, the blood becomes much more diluted, as evidenced by the dramatic reduction in serum albumin levels that occurs during pregnancy Preeclampsia-related disorders that increase the risk of developing pulmonary oedema. It is crucial to remember that pulmonary edema detected in pregnancy often responds substantially to withdrawal of the underlying cause and administration of diuretic, even if it is severe clinically and radiologically [27].

Incidence of preeclampsia[28].
Parity…. first pregnancy.
Age…. young adolescent
Gestational Age…. < 34 weeks.
Racial Factors…. black race
Familial Factors…history.
Genetic Factors…susceptibility genes
Obesity…. more risk factor
Cigarette Smoking…. induced
Previous History…. secondary pregnant increased incidence.
Hyperplacentosis…. twins gestation
Dietary habits… lower socioeconomic classes.

Classification of preeclampsia: -[29]
(A)Mild Preeclampsia Hypertension (SBP ≥ 140 mmHg or DBP ≥ 90 mmHg), may be superimposed on chronic hypertension
Proteinuria (proteinuria ≥ 300 mg/24 hours, or significant increase from baseline)
(B)Severe PreeclampsiaSustained SBP ≥ 160 mmHg or DPB ≥ 110 mmHg (measured twice, at least six hours apart)Evidence of other end-organ damage (if one or more of the following) :
Deteriorating renal function includingproteinuria ≥ 3 g/24 hours or 3+ on urine dipstick or sudden oliguria, especially with elevated creatinine**.**
CNS disturbance (altered vision, headache) and Pulmonary edema (3% of patients)

HELLP syndrome is a life-threatening obstetric complication. Hemolysis, elevated liver enzyme, low platelet. Eclampsia is a complicates 1-2% of cases of severe preeclampsia, is defined as the occurrence of tonic-colonic seizures in a pregnant.

The main goals of treatment in severe preeclampsia are blood pressure control (BP) and prevention of eclampsia, with vaginal delivery for appropriate patients and cesarean section in cases of urgency or when induction labor fails, with timing balanced by the safety of the pregnant mother versus risk of delivery of a potentially premature baby [30].] Antihypertensive drug stability, stable laboratory readings, and a reassuring fetal biophysical profile are all prerequisites for expecting management. Maternal and fetal monitoring should be included as part of expectant management criteria, and delivery should only take place in an advanced-care facility [31].

Organ failure can occur when magnesium sulfate (MgSO4) medication is administered in the intensive care unit. A Glasgow score of 15 is used, as are tendon reflexes, respiratory frequency >12 beats per minute, and

diuresis >30 mL/hour. This monitoring is based on these criteria: As soon as there are signs of an overdose, it's necessary to stop the infusion and consider administering calcium gluconate injections. A serum MgSO4 value of 5–8 mg/dL is the target. Magnesium sulfate can be stopped in patients whose blood pressure (BP) has been successfully stabilized and closely monitored in a high-risk antepartum unit until delivery, when it is restarted. The infusion must be continued for at least 24 hours postpartum because of the ongoing risk of eclampsia [32].

Intraamniotic Infection

If you have an infection in the amniotic fluid and it results in inflammation of the amniotic fluid, placenta, fetus or fetal membranes (or decidua), you have chorioamnionitis (intraamniotic infection). Some writers have proposed renaming this syndrome to "intraamniotic infection and inflammation" to better reflect the entire scope of the disease process [33].

Polymicrobial origins are typical in intraamniotic infection; both aerobic and anaerobic bacteria are usually found; and the flora of the vaginal cavity is frequently the source [34]. When an infection develops in the mother while she is pregnant, the risks to the mother can be severe. These risks include preterm labor requiring more intervention, postpartum uterine atony with hemorrhage and other complications such as infection of the mother or the unborn child, endometritis and peritonitis [35]. High-risk factors for intraamniotic infection during pregnancy include low parity (eg. group B streptococcal infection and sexually transmitted infections), multiple digital examination, use of internal uterine and fetal monitors, meconium-stained amniotic fluid, and sexually transmitted infections (eg. chlamydia pneumoniae) [36].

Amniotic fluid culture, gram stain, or both, as well as a biochemical investigation, can be used to objectively diagnose intraamniotic infection, although for most women in labor at term, the diagnosis is made primarily by clinical criteria [37]. An expert panel of maternal and neonatal experts recently recommended categorizing intraamniotic infection into three different categories: 1) isolated maternal fever, 2) suspected intraamniotic infection, and 3) confirmed intraamniotic infection. The workshop was sponsored by the Eunice Kennedy Shriver National Institute of Child Health and Human Development, the Society for Maternal–Fetal Medicine, and the American Academy of Pediatrics (1). According to clinical and laboratory/pathologic results, the new definitions discriminate between suspected and proven intraamniotic infection; they also give standardized temperature parameters for diagnosing intrapartum fever [38]. Additionally, studies have indicated that intrapartum antibiotics cut down on maternal febrile morbidity and length of stay in the hospital. If intraamniotic infection is suspected or confirmed, use of intrapartum antibiotics is recommended in the absence of any clearly demonstrated overriding hazards [39]. Antipyretics should be used in conjunction with antibiotics to reduce fever. Given the link between intraamniotic infection and a dysfunctional labor progression, proper labor progression should be enforced [40].

Ovarian Hyperstimulation Syndrome [OHSS]

Most occurrences of OHSS are caused by ovaries being exposed to hCG or LH after regulated ovarian stimulation with follicle-stimulating hormone (FSH). When hCG is administered to overstimulated ovaries, proinflammatory mediators are produced. There are numerous cytokines that may be involved in the pathophysiology and clinical manifestations of OHSS, with vascular endothelial growth factor (VEGF) being the most important [41]. This condition's symptoms are caused by the presence of ovarian enlargement in conjunction with the local and systemic effects of proinflammatory mediators, such as enhanced vasodilation and a prothrombotic impact. Fluid loss into third space is caused by an increase in vascular permeability; this is seen most commonly in the form of ascites [42]. Severe hypovolemia in women with OHSS manifests as a typical loss of 20% of their calculated blood volume in the acute phase of OHSS, dyspnea, oliguria, and severely enlarged polycystic ovaries. Other symptoms include hemoconcentration, a hematocrit greater than 55%, a WBC count greater than 25,000, and thromboembolic phenomena. Failure of the kidneys [43].

Hypovolemia is accompanied by a drop in serum osmolality and salt concentrations. A'reset' of the osmotic thresholds between thirst and vasopressin to reduce osmolality and sodium levels is thought to be the cause of this paradoxical combination of hypovolemia and hypo-osmolality in these women. The observed declines in serum osmolality and sodium can be attributed to the simultaneous resetting of the osmotic thresholds rather than electrolyte losses [44].

OHSS is diagnosed by a clinical process. After the trigger injection used to encourage final follicular maturation before oocyte retrieval, the average patient has abdominal distension and discomfort. However, the absence of an extensive previous ovarian response to stimulation does not exclude the diagnosis of OHS [45]. Patients are divided into two categories based on when symptoms appear after receiving a trigger injection: those with early OHSS and those with late OHSS. This type of OHSS usually appears within seven days of the hCG injection and is linked to an overactive ovarian response.

Early OHSS Endogenous HCG from an early pregnancy might cause 'late'OHSS, which shows up 10 days or more after the hCG injection. These women's prior ovarian response may be ordinary. The late stage of OHSS is more severe and lasts longer than the early stage [46].The syndrome is self-limiting and resolves within 10–20 days in pregnant women, while HCG levels are declining. The initial step in managing these patients is to evaluate their hemodynamic and respiratory condition. Monitoring of complete blood counts, electrolytes, and renal and hepatic functions in the laboratory. Monitoring. Fluid management may be necessary if the condition is severe (i.e., CVC is preferable) [47]. Close monitoring of the urine output with the Foley's catheter is recommended. Crystalloid is the preferred crystalloid. either glucose or plain old normal saline If your albumin level is below 3.0 g/dL, you should take an albumin plasma expander and diuretics (furosemide) to treat your hypoalbuminemia. Thoracentesis/paracentesis should be performed if respiratory symptoms worsen. Mechanical ventilation may be required if ARDS develops, Lung-protective techniques must be adopted in this situation [48].

III. CONCLUSION

The morbidity and mortality associated with this catastrophic consequence must be minimized by a multidisciplinary team of physicians for pregnant patients with acute respiratory failure (ARF) who are admitted to the ICU. A condition known as peripartum cardiomyopathy (PPCM) occurs in the final month of pregnancy and continues into the fifth month after childbirth. It is an idiopathic dilated cardiomyopathy. Heart failure must have no other known cause, and there must be no known heart illness before the last month of pregnancy. Although amniotic fluid embolism (AFE) is a rare occurrence, it can have devastating consequences for the unborn baby. The cause is unknown, however amniotic fluid containing vernix and other substances entering the mother's circulation is likely to be a factor. Anaphylaxis-induced cardiorespiratory collapse, altered mental status, and DIC are all possible outcomes of amniotic fluid and fetal debris entering the systemic maternal circulation during an AFE. There are two types of preeclampsia: mild and severe. Mild cases occur before 20 weeks of pregnancy, whereas severe cases can appear as late as 4-6 weeks after delivery. Patients with this condition have hypertension, proteinuria, and pathologic edema.

The clinical symptoms of OHSS are caused by the ovarian hyper stimulation syndrome (OHSS), which is characterized by ovarian enlargement accompanied by the local and systemic effects of proinflammatory mediators, such as increased vascular permeability and a prothrombotic effect. Fluid loss into the third space is a result of increased vascular permeability, which can show up as ascites or less usually as pleural and pericardial effusions. Acute hypovolemia is seen in women with severe OHSS, with a typical 20 percent reduction of their estimated blood volume. Tocolytic therapy-induced pulmonary edema usually happens when the medicine is taken orally or intravenously, or within 24 hours of stopping the beta-agonist medication. When tocolytic medication fails to stop labor, it's possible that respiratory problems will arise during the first 12 hours after delivery. Multiple pregnancies or concurrent conditions such as localized or systemic infections, silent heart disease, or treatment with magnesium sulfate or corticosteroids place patients at greater risk for this problem.
From this study it is concluded that the intensivist must be aware of: -

. different causes of respiratory failure during pregnancy.
. differential diagnosis and management of these patients.

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