REVIEW ARTICLE

Postpartum Acute Pulmonary Edema

Pranav Sawant, Shweta Deshmukh, Jitendra Ingole

Department of Medicine, SKNMC and GH, Pune, Maharashtra, India

ABSTRACT

It is uncommon for women to experience acute dyspnea after giving birth, and it may be accompanied by a number of serious illnesses. Pneumonia, aspiration, pulmonary edema, pulmonary embolism, and amniotic fluid embolism are the most likely reasons that need to be taken into account. About 0.08–1.5% of women experience acute pulmonary edema (APO) of postpartum, which is a medical emergency. Pregnant women who have APO have higher rates of maternal morbidity and mortality. This article gives a general review of APO in postpartum women, including typical causes, difficulties in diagnosis and research, and management and treatment options.

Key words: Acute dyspnea, Postpartum, Pulmonary edema

INTRODUCTION

The main cardiovascular complication of preeclampsia, acute pulmonary edema (APO),[1] which occurs in 3% of patients and 70% of which occur during postpartum, is one of the criteria for severe preeclampsia. APO in preeclampsia is primarily caused by advanced maternal age, multiple pregnancies, and persistent hypertension.

Pulmonary edema following childbirth is a rare clinical condition.[2] The fourth most frequent type of maternal morbidity and a main cause of death in preeclamptic women is APO, a symptom of severe illness. It can happen during the prenatal, intrapartum, or postpartum periods[3] and is frequently the cause of intensive care hospitalization. Around 0.05% of low-risk pregnancies are complicated by pulmonary edema, but preeclampsia-complex pregnancies are up to 2.9% more likely to experience it, with 70% of cases developing after birth.[3-5] A clinician needs to be aware of how pregnancy-related physiological changes to the mother's cardiovascular system, such as increases in plasma blood volume, cardiac output, heart rate, and capillary permeability and decreases in plasma colloid osmotic pressure, predispose to the development of pulmonary edema. The first goal is resuscitation, next comes the creation of a differential diagnosis to address the underlying illness [Table 1].

Access this article online	
Quick Response code	**Website:** www.mjbar.in
	Recevied on: 23/12/2022 **Accepted on:** 17/01/2023

CAUSES AND DIFFERENTIAL DIAGNOSIS

An abrupt rise in cardiac output, which may not be tolerated by people with underlying cardiac illness and results in decompensated heart failure, is one of the physiological changes that occur in the early postpartum period. RHD is one of the most common cardiovascular illnesses that affect pregnant women, especially in underdeveloped countries, and it causes substantial morbidity.[5]

The use of tocolytic medications, underlying heart illness, fluid overload, and preeclampsia are the most frequent causes of pulmonary edema [Figure 1].[2]

PATHOPHYSIOLOGY

A complex interaction between elevated intravascular hydrostatic pressures, increased vascular permeability, and decreased intravascular colloid osmotic pressures, particularly in preeclampsia, contributes to the predisposition of pulmonary edema in postpartum pregnancy (38%) and causes fluid extravasation into the pulmonary interstitium.[6]

Address for correspondence:
Dr. Pranav Sawant, Department of Medicine, SKNMC and GH, Pune, Maharashtra, India. Tel.: 7507730911.
E-mail: pranavsawy@gmail.com

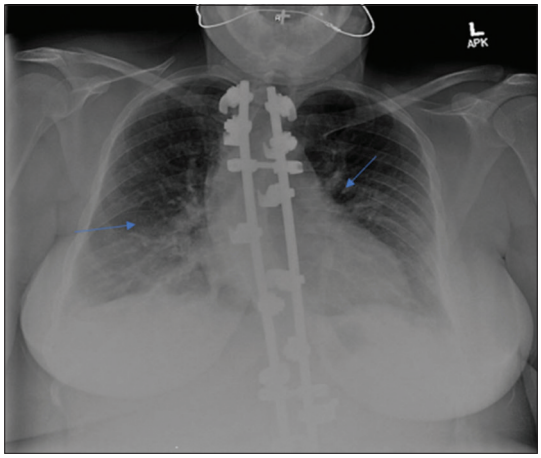

Figure 1: Chest X-ray showing pulmonary edema with bilateral vascular congestion with mild interstitial thickening (arrows)[6]

INVESTIGATIONS AND TREATMENT

Due to the intricate nature of the changes that take place during pregnancy, women are more vulnerable to a wide range of diseases. Pregnancy symptoms that are frequently experienced by women include frequent urination, exhaustion, poor sleep, nausea, vomiting, back pain, and pelvic discomfort.[2,3] In addition, hypertension-related diseases, such as chronic hypertension, gestational hypertension, and preeclampsia, complicate 5–10% of pregnancies.[4] The resolution of pregnancy symptoms and return to the non-pregnant condition is usually brought on by the delivery of the fetus and placenta. The most frequent etiological cause causing APO was HDP. The most frequent factor contributing to pulmonary edema in preeclampsia with an early onset was hypertension. Cardiovascular problems with valvular heart

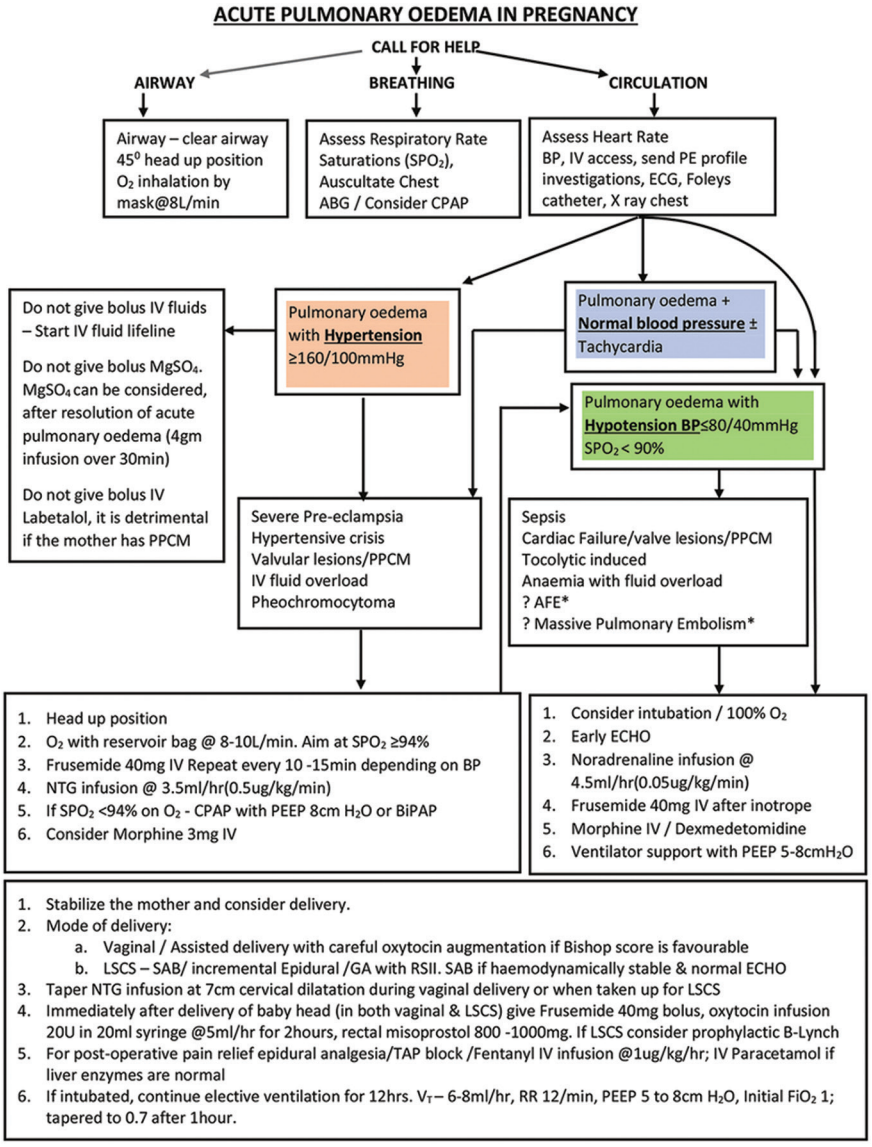

Figure 2: Acute pulmonary edema protocol.[10]

Table 1: Differential diagnoses of acute dyspnea in the postpartum patient stratified by pulmonary edema versus non-pulmonary edema causes[6]

Without Pulmonary edema	With Pulmonary Edema	Non-Cardiogenic
	Cardiogenic	
Pulmonary Embolism		Letrogenic fluid overload
Amniotic fluid embolism	Peripartum Cardiomyopathy	Thyroid diseases
Pneumonia	Preeclampsia-related heart failure	Tocolytic therapy
Foreign body dyspnea	Underlying cardiac diseases (e.g- Vulvulopathy)	Medication-related sepsis
Psychogenic dyspnea	Myocardial ischemia	Acute respiratory distress syndrome
	Sepsis with poor cardiac output	

illnesses and cardiomyopathy were the second most frequent etiology. While sepsis and severe anemia were the major risk variables, multiparity was related with a decreased incidence of pulmonary edema.[7]

The main factor is preeclampsia, which is followed by heart issues, tocolysis, and overfilling. One of the potential causes that can be prevented is the intravenous use of beta-agonists.[7] A Doppler echocardiography is the examination of choice for suspected pregnant individuals to discover subclinical valvular heart disease and to prevent unfavorable consequences during the post-operative phase. Finding underlying heart disease in patients with acute pulmonary edema is critical. Remote places of the world should have access to portable and transportable echocardiography.[8]

TREATMENT ALGORITHM

Patients who had been admitted with shortness of breath (SOB) and clinical signs of pulmonary edema underwent pulse oximetry monitoring, clinical respiratory rate counting, and non-invasive blood pressure monitoring.[9] The presence of SOB, an elevated respiratory rate of more than 35 breaths/min, a drop in saturations to <94% on room air, and bilateral crepitations on pulmonary auscultation were used to make the diagnosis of APO. Following the establishment of the clinical diagnosis, the management was established in accordance with the APO hospital policy [Figure 2].[10]

Every 15 min, the patients were evaluated for clinical improvement. Increased saturations were viewed as a successful outcome. Clinical stabilization was indicated by an increase in saturations to over 94% and a reduction in respiratory rate to under 35 breaths per minute. It was documented what was being treated and when the patient first stabilized. Delivery method, anesthesia used, any morbidity or mortality, and the result for the newborn were all reported. To investigate changes between pre- and post-protocol periods, a statistical analysis was conducted. Point estimates and their 95% confidence intervals were calculated. *P*-value was calculated using Fisher's exact test for categorical variables and Student's t test for continuous variables. $P = 0.05$ was considered statistically significant.[10]

CONCLUSION

Although it is uncommon for postpartum individuals to come with acute dyspnea, a comprehensive workup is necessary due to the possibility of a high-risk condition with a high mortality rate. Therefore, it is essential to do strict monitoring for all illnesses, including peripartum cardiomyopathy, postpartum preeclampsia, pulmonary embolism, and other causes of postpartum flash pulmonary edema, when a postpartum patient with dyspnea symptoms is admitted. As part of the initial workup, a complete examination should be performed that includes a stat echocardiography, a chest X-ray, an EKG, a urinalysis, and repeated blood pressure readings. The majority of postpartum pulmonary edema treatment is supportive, including blood pressure management, diuretics, and, where necessary, breathing support.

TAKE HOME POINTS

1. In cases of APO without hypertension, iatrogenic causes continue to be a significant factor. APO has been linked to the therapy of premature labor using tocolytic drugs like adrenoceptor antagonists (terbutaline and salbutamol). Both corticosteroids and magnesium sulfate have been linked to the development of APO in pregnant or postpartum women. The development of APO is known to be significantly increased by unrestricted intravenous fluid treatment
2. Measures to prevent additional complications include cesarean sections, magnesium sulfate seizure prophylaxis, restrictions on fluids for laboring women, deep vein thrombosis, pulmonary embolism, and stress ulceration of the gastrointestinal system
3. Early intervention by multidisciplinary teams involvement and communication, close monitoring and recording of observations (including conscious state, blood pressure, respiratory rate, heart rate, oxygen saturation, temperature, and fluid balance), avoidance of

non-steroidal anti-inflammatory drugs, restricted fluid administration, and minimization of add are strategies to reduce the risk of APO in pregnant women. Transthoracic echocardiography can help distinguish between low and high cardiac output states and rule out other significant causes of APO. It should be obligatory to monitor electrocardiography, blood pressure, oxygen saturation, heart rate, respiration rate, temperature, and fluid balance.

REFERENCES

1. Shadrach BJ, Tiwari D, Shahi SS, Goel R. Postpartum Cardiogenic Pulmonary Edema: The initial presentation of an underlying rheumatic heart disease. Indian J Crit Care Med 2020;24:999-1000.
2. Sciscione AC, Ivester T, Largoza M, Manley J, Shlossman P, Colmorgen GH. Acute pulmonary edema in pregnancy. Obstet Gynecol 2003;101:511-5.
3. Pordeus AC, Katz L, Soares MC, Maia SB, Amorim MM. Acute pulmonary edema in an obstetric intensive care unit: A case series study. Medicine (Baltimore) 2018;97:e11508.
4. Dunne C, Meriano A. Acute postpartum pulmonary edema in a 23-year-old woman 5 days after cesarean delivery. CJEM 2009;11:178-81.
5. Soma-Pillay P, Nelson-Piercy C, Tolppanen H, Mebazaa A. Physiological changes in pregnancy. Cardiovasc J Afr 2016;27:89-94.
6. Randhawa JS, Ashraf H, Colombo J, Kudla P. Postpartum respiratory distress due to hypertension-related pulmonary edema. Cureus 2021;13:e18179.
7. Padmaja R, Rao SK. Gande: Postpartum acute pulmonary oedema with sub clinical rheumatic heart disease. J Clin Dign Res 2015;9:UD01-2.
8. Dennis AT, Solnordal CB. Acute pulmonary oedema in pregnant women. Anaesthesia 2012;67:646-59.
9. Kousalya C, Swetha T, Praveen NK, Anuradha A, Nagamani S. Protocol-based management of acute pulmonary edema in pregnancy in a low-resource center. J Obstetr Anaesth Crit Care 2020;10:98.
10. Dolley P, Lebon A, Beucher G, Simonet T, Herlicoviez M, Dreyfus M. Acute pulmonary edema and pregnancy: A descriptive study of 15 cases and review of the literature. J Gynecol Obstet Biol Reprod (Paris) 2012;41:638-44.

How to cite this article: Sawant P, Deshmukh S, Ingole J. Postpartum Acute Pulmonary Edema. Med J Basic Appl Res 2023; 4(1):29-32.

Conflicts of Interest: None. **Source of support:** None.