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CASE STUDY

Severe Respiratory Distress after C Section Due to Hypertension-Related Pulmonary Edema : A Case Report

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ABSTRACT

Background: Pulmonary edema is a very rare complication of pregnancy. It is more commonly seen as a complication of preeclampsia. The physiopathology of this relationship is not well understood. This is a life-threatening condition that requires immediate treatment and termination of pregnancy.

Case Presentation: We present the case of a women 24 year old who developed preeclampsia, complicated by pulmonary edema (pe) with heart failure after elective c section under spinal anesthesia in her first pregnancy. In the Intensive Care Unit (ICU) the patient received fist using Non-invasive ventilation (niv), after six hour using niv the oxygen saturation go down, and move using ventilator intubation, using ARDS NET hight peep.

Discussion: Acute respiratory distress syndrome (ARDS) is a life threatening condition characterized by poor oxygenation and noncompliant or "stiff" lungs. This disorder is associated with capillary endothelial injury and diffuse alveolar damage. Many mechanisms have been proposed to explain the pathogenesis of pulmonary oedema in pre-eclampsia including hypervolaemia, left ventricular failure and pulmonary capillary leakage.

Conclusion: Pulmonary edema is an emergency that can occur in pregnancy with preeclasmsia which has a high mortality if not treated immediately. Early screening and adequate therapy can improve patient outcomes.

Keywords : Severe Respiratory Distress, Hypertension, Pulmonary Edema

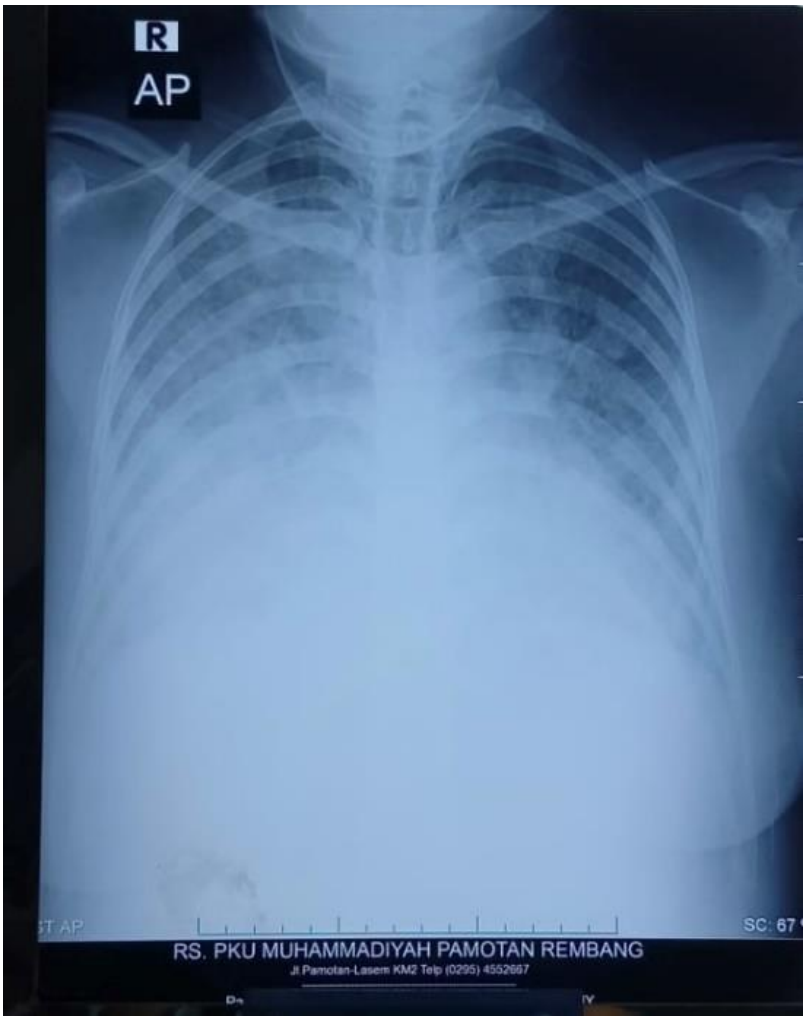
Background

Pulmonary edema is a very rare complication of pregnancy. It is more commonly seen as a complication of preeclampsia. The physiopathology of this relationship is not well understood. This is a life-threatening condition that requires immediate treatment and termination of pregnancy [1]. In the postpartum condition the body will immediately engage in the largest cardiac output during pregnancy due to increased venous return due to loss of compression of the vena cava and movement of blood from the empty and contracted uterus into the systemic circulation. Although well tolerated in most pregnancies, the increased hemodynamic burden in the postpartum period can lead to heart failure in women with cardiac disease. Acute pulmonary edema in patients with coronary artery disease is a dramatic manifestation of heart failure. It has recently been recognized that patients with acute pulmonary edema often have hypertension and their systolic function is preserved even after coronary revascularization. More than 90% of patients suffering from heart failure have diastolic dysfunction regardless of left ventricular ejection fraction (LVEF). These findings suggest that diastolic dysfunction is an important contributor to acute hypertensive pulmonary edema in patients with coronary artery disease [2].

We report a single case of a 24 year old woman who developed preeclampsia, complicated by pulmonary edema with heart failure after elective caesarean section under epidural anesthesia in her first pregnancy. Acute respiratory distress syndrome (ARDS) is a life-threatening condition characterized by poor oxygenation and noncompliant or "stiff" lungs. This disorder is associated with capillary endothelial injury and diffuse alveolar damage [3]. The resultant disruption of the alveolar epithelial endothelial barrier results in accumulation of a protein-rich pulmonary oedema, surfactant dysfunction, and impaired gas exchange [11]. When ARDS develops, patients usually experience varying degrees of pulmonary artery vasoconstriction and may develop pulmonary hypertension. ARDS has a high mortality rate, and few therapeutic modalities are effective in alleviating this deadly condition [3].

Case Presentation

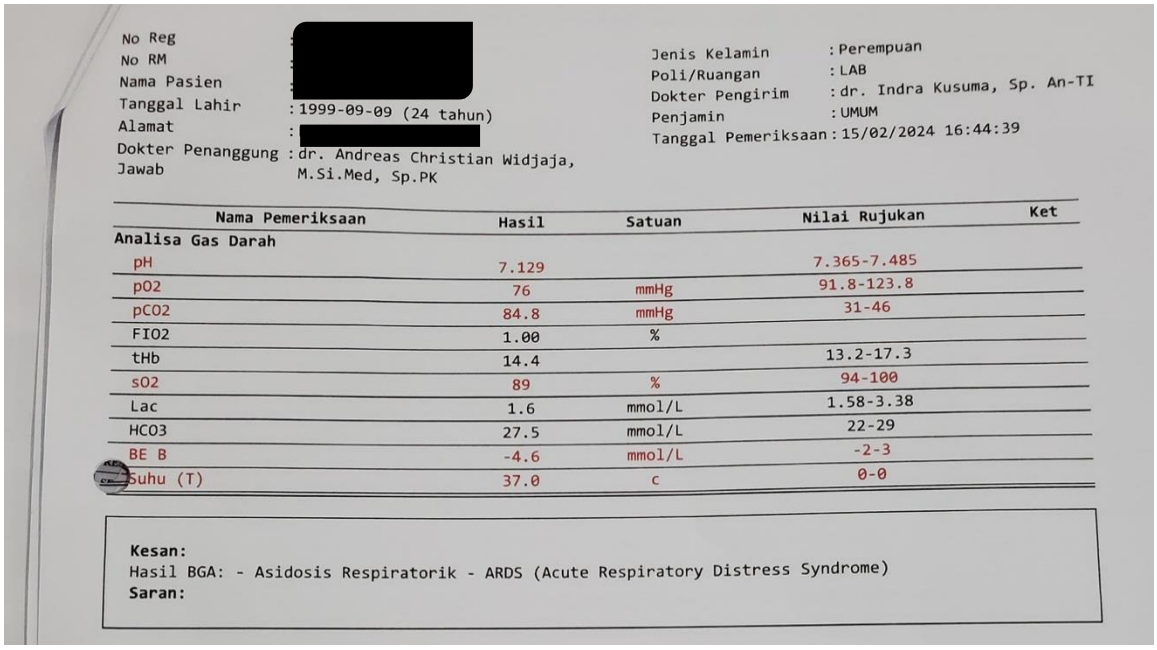
A woman with a first pregnancy aged 38 weeks with preeclampsia and cpd. The patient arrived at the Emergency Room (ER) with a blood pressure of 130/90 without any nausea, shortness of breath or dizziness. The patient was then planned for section using moderate anesthesia with spinal anesthesia technique, with stable blood pressure monitoring of 120/90, respiratory rate (rr) 20, heart rate (hr) 90, and Spo2 99-100%. After the cesarean section was carried out and the patient was moved to the recovery room, the patient experienced shortness of breath with rhonchi + on examination of the right lung. The patient experienced shortness of breath 3 hours after the cesarean section, with saturation 88%, (rr) 50, (hr)163, blood pressure 230/99. The patient was then transferred to the intensive care unit where a thorax x-ray (figure 1.1) was performed and echo with an ejaculation fraction result of 42%. In the Intensive Care Unit (ICU) the patient received fist using Non-invasive ventilation (niv), after six hour using niv the oxygen saturation go down, and move using ventilator using ARDS NET hight peep, by carrying out suction through the connector and there was positive frosty pinky sputum (figure 1.2).



(figure 1.1) Chest X-ray showing pulmonary edema

(figure 1.2) Frothy pink sputum with furusemide 10 mg syringe therapy pump, NTG Syring Pump (SP) titration starting at 20 micro/hour, knock down morfin 1mg/ hour and midazolam 2 mg/hour, infusion 250cc/24 hours, levofloxacin 750mg/24 hours, azetazolamide 3x1, spironolactone 25mg 1x1, digoxin 1x0.25mg, omeprazole 40mg /12 hours, paracetamol 1 gram/ 8 hours intravenously.

A complete blood examination and Blood Gas Analysis (BGA) showed the following results, pCO2 84.8, pO2 76, HCo3 27.5, pH 7.129 with the interpretation of respiratory acidosis (figure 1.3).



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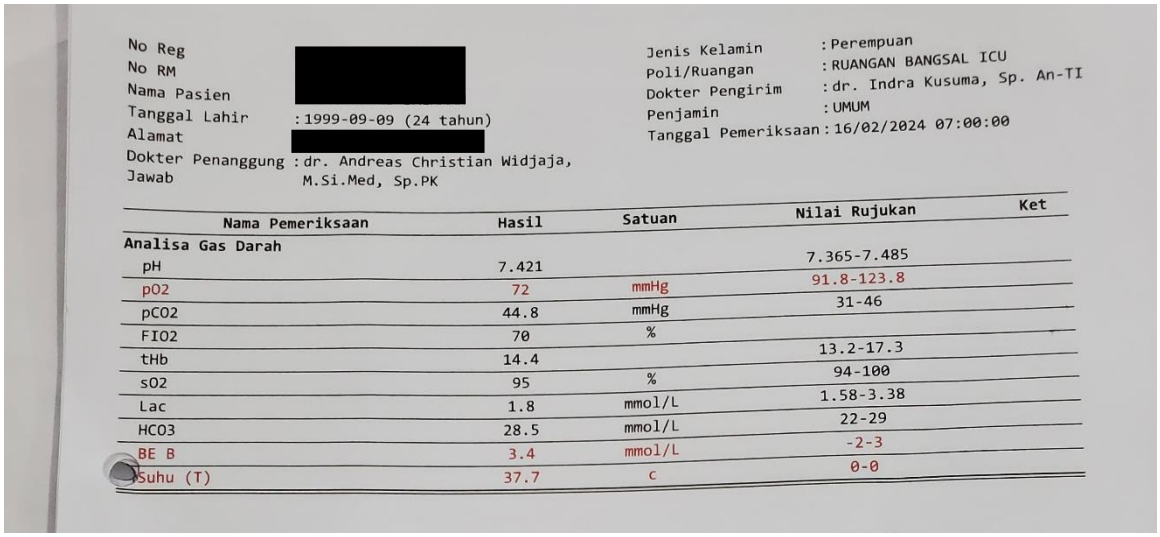
Poli/Ruangan : LAB
Dokter Pengirim : dr. Indra Kusuma, Sp. An-TI
Penjamin : UMUM
Tanggal Pemeriksaan : 15/02/2024 16:44:39

Nama Pemeriksaan	Hasil	Satuan	Nilai Rujukan	Ket
Analisa Gas Darah				
pH	7.129		7.365-7.485	
pO2	76	mmHg	91.8-123.8	
pCO2	84.8	mmHg	31-46	
FIO2	1.00	%		
tHb	14.4		13.2-17.3	
sO2	89	%	94-100	
Lac	1.6	mmol/L	1.58-3.38	
HCO3	27.5	mmol/L	22-29	
BE B	-4.6	mmol/L	-2-3	
Suhu (T)	37.0	c	0-0	

Kesan:
Hasil BGA: - Asidosis Respiratorik - ARDS (Acute Respiratory Distress Syndrome)
Saran:

(Figure 1.3) Blood gas analysis before treatment in ICU.

With the addition of etambiol 3x1 and vitamin A 1x1. On the following day after therapy the patient's shortness of breath had decreased with the results of vital signs blood pressure 106/70, heart rate (hr) 108, respiratory rate (rr) 22, SpO2 97% and continued therapy ventilator began to decrease gradually according to ARDS NET, nebulizer combivent + budesma 1 respul/ 8 hours, NAC 3x1, fentanyl starts to decrease by 25 micro after stopping, midazolam / hours, extra injection of metamizole 1 gram, follow-up therapy for NTG starts to decrease by 5 mcg/min after stopping, furosemide 5



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Tanggal Pemeriksaan : 16/02/2024 07:00:00

Nama Pemeriksaan	Hasil	Satuan	Nilai Rujukan	Ket
Analisa Gas Darah				
pH	7.421		7.365-7.485	
pO2	72	mmHg	91.8-123.8	
pCO2	44.8	mmHg	31-46	
FIO2	70	%		
tHb	14.4		13.2-17.3	
sO2	95	%	94-100	
Lac	1.8	mmol/L	1.58-3.38	
HCO3	28.5	mmol/L	22-29	
BE B	3.4	mmol/L	-2-3	
Suhu (T)	37.7	c	0-0	

mg/hour. Blood Gas Analysis after that treatment the result of BGA pCO2 44.8, pO2 72, HCo3 28.5, pH 7.421 (figure 1.4).

(Figure 1.4) Blood gas analysis after treatment in ICU.

After treatment in the ICU for 4 days the patient began to cooperate with the ventilator installed in CPAP mode, the patient's consciousness was compos mentis, gcs e4 Vet m6 blood pressure 96/59, rr 24, hr 100, spo2 98% with continued therapy head up 30-40 degrees , section if necessary, diet nefrisol 4x200cc, sp fentanyl if finished stop, syring pump (sp) fufosemide 2.5 mg/hour, sp miloz 0.5/hour if finished stop, sp NE 0.025 mcg/kgbb/min, nebulaizer combivent + budesma / 6 hours, peep decreases 1 every hour until 5, p decreases gradually target until o every 1 hour 1, if stable V:E >300 extubation plan and score this patients zero from seven criteria extubation. On the 6th day after extubation the patient did not complain of shortness of breath, had a mild cough, vision was not blurred with compos mentis awareness and blood pressure results were 114/76, hr 88, rr 20, spo2 97%, with frutrolit infusion therapy 250cc/24 hours, inj. Furosemide/24 hours, Nebulizer Combivent + Budesma / 8 hours, injection Bromax 1x1, O2 released with a minimum saturation target of 88-95%, breathing exercise chest therapy. The patient was planned to go home with oral therapy of furosemide 1x20mg, spironolactone 1x25mg, digoxin 1x0.25mg, concor 1x1.25mg, captopril 2x12.5 mg.

Discussion

Pregnancy can cause many changes to the cardio vascular system. These changes include changes in cardiac output, heart rate, blood pressure, vascular resistance, ventricular capacity and size. The mother will experience an increase in heart rate of 10 to 20 times per minute at the beginning of the third trimester. By 28 weeks, the maximum fetal growth rate has occurred, and the uterine tissue growth slows while continuing to stretch rapidly and become thin. Within several weeks of delivery, the uterus then returns to its pre-pregnancy structure [10]. Early-onset hypertension and/or preeclampsia occurs before 34 weeks of gestation and is attributed to syncytiotrophoblast stress leading to poor placentation, whereas late-onset hypertension and/or preeclampsia occurs at 34 weeks onward and is thought to be secondary to the placenta outgrowing [7,12,13]. Along with an increase in stroke volume of 25%, resulting in an overall increase in cardiac output of 50%. The increase in cardiac output is a compensation due to an increase in heart rate, a decrease in vascular resistance and an increase in stroke volume. The decrease in systemic vascular resistance continues until term, this is caused by the hormone progesterone which causes relaxation of smooth muscles and has an impact on vasodilation in blood vessels. During labor and the postpartum period there will be an increase in cardiac output, where an increase of 12% occurs in the first stage of labor and is also the main cause of increased stroke volume. This increase is thought to occur due to increased preload during each uterine contraction, this phenomenon is often referred to as "uterine autotransfusion". During the second stage, cardiac output can increase up to 34%, which initially starts with an increase in stroke volume, followed by an increase in cardiac output and is characterized by an increase in heart rate. Immediately after delivery (10-30 minutes after the baby is born) it is estimated that 300-500 mL of blood that previously flowed to the uterus will return to the mother's vascular circulation. This increase will play a role in increasing preload and stroke

volume and will then also increase cardiac output by 10-20%. This incident should be of particular concern to patients who have a history of heart disease, because it can cause pulmonary edema or congestive heart attack [4]. The physiologic changes associated with pregnancy, including an increase in intravascular volume (preload), may create a predisposition to acute pulmonary edema. Specifically, a hypertensive crisis (afterload) may cause a severe increase in pulmonary capillary pressure, resulting in alveolar hemorrhage. Flash pulmonary edema is associated with a sudden rise in left-sided intracardiac filling pressures in settings such as a hypertensive emergency [5]. Many mechanisms have been proposed to explain the pathogenesis of pulmonary oedema in severe pre-eclampsia including hypervolaemia, left ventricular failure and pulmonary capillary leakage. Pulmonary oedema could be due to a combination of these factors. However, it is thought that increased systemic vascular resistance induces significant changes in loading conditions of the ventricular myocardium contributing to diastolic filling abnormalities and to the development of an ischemic substrate with the potential for development of heart failure and pulmonary edema [1]. The treatment strategy is supportive care, focusing on reducing shunt fraction, increasing oxygen delivery, decreasing oxygen consumption, and avoiding further injury. Patients are mechanically ventilated, guarded against fluid overload with diuretics, and given nutritional support until improvement is observed [9]. Interestingly, the mode in which a patient is ventilated affects lung recovery. A lung-protective ventilatory strategy is advocated to reduce lung injury [6]. Several studies have reported that weaning protocols reduced the total duration of ventilation, weaning duration, and intensive care unit (ICU). Meanwhile, the extubation process is a critical compo- nent of respiratory care in patients who receive mechan- ical ventilation. First risk assessment checklist tolerance for spontaneous breathing trial (SBT), the patient eligibility for extubation if they do not meet at leat one of this seven criteria. (1) Respiratory rate (rr) >35 mn for 5 minutes or more, (2) Rapid shallow breathing index (RSBI) >100 cysles/min/L, (3) SaO2<95% for 5 minutes or more, (4) Heart rate >120/min or sustained increase 20% greater than baseline, (5) Systolic blood pressure <90 mmhg or >180 mmh for 5 minutes or more, (6) emergence or a chest pain or a new electrocardiogram change, (7) Dyspnea, increased anxiety and diaphoresis. If they meet at leat one of these seven criteria, they are rested on mechanical ventilation until the next morning and rechecked the next day. Second risk assessment checklist eligibility for extubation, (1) no severe disturbance in level of consciousness e.g ability to protect the airway, (2) presence of cough reflex, (3) presence of gag reflex, (4) stable cardiovascular system e.g Hr <120 beat/min, no ischemic change on electrocardiogram and no severe cardiac arrhythmia, (5) rr <35 breath / min , (6) RSBI <100 breaths/min/L, (7) presence of cuff leak. Patients are extubated if they satisfy all of the above criteria. If the do not mechanical ventilation is continued , and items in this checklist are rechecked the next day [8].

Conclusion

In physiological pregnancy, the body will increase the total volume of fluid, including changes in cardiac output, heart rate, blood pressure, vascular resistance, capacity and size of the ventricles, with the presence of risk factors for heart failure making pregnancy susceptible to acute lung edema. Pulmonary edema is an emergency that can occur in

pregnancy with preeclasmsia which has a high mortality if not treated immediately. Early screening and adequate therapy can improve patient outcomes.

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