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Cesarean Section in a Patient with Severe Preeclampsia with Pulmonary Edema: A Case Report

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Abstract: Acute pulmonary edema is a leading cause of death in patients with preeclampsia. The authors reported a case of a pregnant woman at 25 weeks of gestation with severe preeclampsia complicated by pulmonary edema, who required an emergency cesarean section, posing a significant challenge to the anesthesiologist. The patient had developed Type 1 respiratory failure and needed supplemental oxygen with high-flow nasal oxygen. Due to contraindications for neuraxial anesthesia, the cesarean section was performed under general anesthesia. After induction of anesthesia, the patient's hypoxemia worsened. Eventually, after treatment with fluid restriction, diuretics, and albumin, oxygenation improved gradually, and the procedure was performed successfully. Both the patient and the newborn had a good prognosis.

Keywords: Preeclampsia; Pulmonary edema; Cesarean section; Anesthesia

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1. Case information

A 41-year-old female, primigravida at 24 weeks and 6 days of gestation, presented with a 3-day history of epigastric pain and a 1-day history of right upper quadrant pain accompanied by elevated blood pressure. The epigastric pain was unrelated to food intake and progressively worsened. On examination, her blood pressure was 176/111 mmHg, heart rate 77 bpm, respiratory rate 18 breaths per minute, and oxygen saturation (SpO_2) 100% on room air.

The patient had a history of hypertension diagnosed two years prior, which was managed irregularly. She also had hypothyroidism treated with levothyroxine, obesity (BMI 38.6 kg/m²), gastroesophageal reflux disease, migraines treated with amitriptyline, depression-controlled pre-pregnancy but untreated during pregnancy, polycystic ovary syndrome, and a history of laparoscopic ovarian cystectomy. She reported frequent air travel over the previous three weeks.

Laboratory findings revealed significant proteinuria (++++) and the presence of ketone bodies (+). Liver

function tests showed elevated ALT (53 U/L) and AST (38 U/L), with total protein at 60 g/L and albumin at 35 g/L. Other blood tests, including renal function, electrolytes, and coagulation profiles, were unremarkable. Ultrasound findings included fatty liver, and a fetal ultrasound estimated the gestational age to be approximately 23 weeks and 5 days.

The patient was diagnosed with preeclampsia with mild liver dysfunction. She was treated with labetalol (100 mg orally every 8 hours) for blood pressure control and aluminum magnesium carbonate tablets for symptomatic relief. After her abdominal pain improved and her blood pressure was controlled below 140/90 mmHg, she was discharged. A follow-up 24-hour urine protein quantification revealed proteinuria of 3.66 g.

Four days later, the patient was readmitted with epigastric and chest pain radiating to the right shoulder and back, along with shortness of breath. On arrival, her blood pressure was 177/102 mmHg, heart rate 71 bpm, respiratory rate 17 breaths per minute, and SpO_2 98% on room air. On examination, her BP was 195/87 mmHg, SpO_2 96% on 3 L/min nasal oxygen, and fetal heart rate was 148 bpm. She had facial and limb edema and bilateral lung crackles.

She was diagnosed with severe preeclampsia and treated with magnesium sulfate for seizure prophylaxis. Blood pressure was managed with oral nifedipine and intravenous phentolamine. Her symptoms worsened, with new-onset dyspnea, headache, and visual disturbances. Despite oxygen therapy, her SpO_2 remained low, requiring high-flow nasal oxygen at 50 L/min with an FiO_2 of 70%.

Laboratory investigations showed significant proteinuria (+++), hypoalbuminemia (31 g/L), elevated D-dimer (6552 ng/mL), and NT-proBNP (1275 pg/mL). Arterial blood gas analysis revealed Type I respiratory failure. Chest computed tomography angiography ruled out pulmonary embolism but showed bilateral pulmonary infiltrates, pleural effusion, and lower lobe atelectasis. Echocardiography revealed mild mitral regurgitation with normal pulmonary artery pressure and a left ventricular ejection fraction of 65%.

The patient was diagnosed with pulmonary edema. She was treated with diuretics and fluid restriction. Despite blood pressure control within the range of 120–145/70–90 mmHg, her hypoxemia persisted. Multidisciplinary discussions concluded that the patient had chronic hypertension with early-onset severe preeclampsia complicated by pulmonary edema, respiratory failure, and fetal growth restriction. Given the poor maternal and fetal prognosis, urgent preparation for cesarean delivery was recommended to terminate the pregnancy.

2. Anesthesia management

2.1. Preoperative assessment

The patient presented with anxiety, semi-recumbent posture, and an inability to lie flat. She was receiving high-flow nasal oxygen (HFNO) at 50 L/min with a FiO_2 of 70%. Her blood pressure was 140/85 mmHg, heart rate 96 bpm, respiratory rate 16/min, and SpO_2 95%. Physical examination revealed facial and limb edema with bilateral lung crackles. Airway evaluation indicated obesity, a short neck, and a Mallampati grade III classification, suggesting a potentially difficult airway (**Figure 1**). She was classified as ASA III-IV/E by the American Society of Anesthesiologists. The patient had a functional classification of New York Heart Association (NYHA) class III with an activity tolerance of < 4 METs. Her preoperative fluid intake was 1,840 mL, with a urine output of 1,750 mL. Due to the recent administration of low-molecular-weight heparin within 24 hours, neuraxial anesthesia was contraindicated, and a cesarean section under general anesthesia was planned.

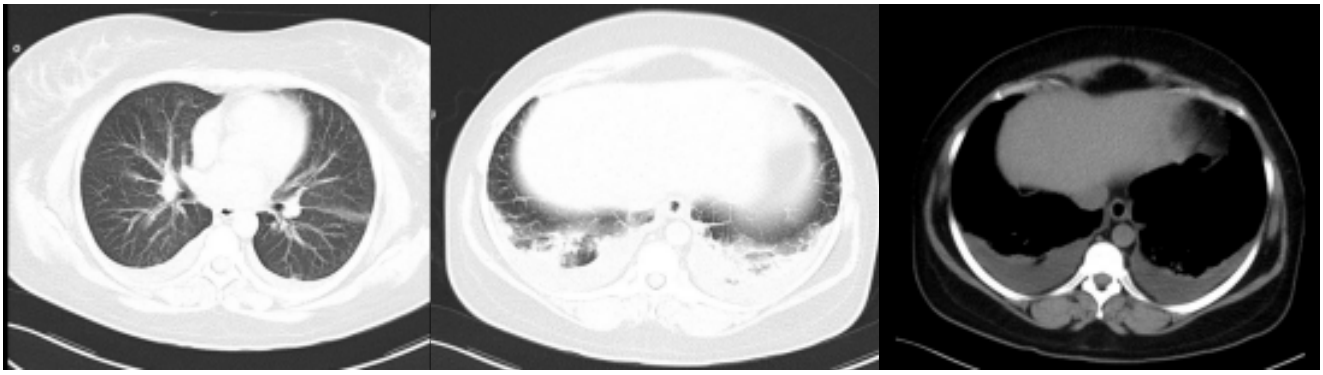

Figure 1. Preoperative pulmonary CTA

2.2. Anesthesia course

Thirty minutes before induction, the patient received intravenous famotidine (20 mg) and metoclopramide (10 mg) to suppress gastric acid and enhance gastric emptying. During transport, she was semi-recumbent, with oxygen provided at 15 L/min by face mask, though she reported dyspnea and restlessness. Upon entering the operating room, oxygen support was continued at the same rate. Initial readings showed BP 191/98 mmHg, HR 96 bpm, RR 20/min, and SpO_2 90–92%, possibly due to HFNO discontinuation, worsening dyspnea, and agitation.

Local anesthesia was administered for a right radial arterial line. Parameters such as arterial blood pressure (ABP), cardiac output (CO), stroke volume variation (SVV), and bispectral index (BIS) were monitored. Concurrently, surgical preparation began.

After 15 minutes, rapid sequence induction was performed using etomidate (16 mg), remifentanil (100 μg), succinylcholine (100 mg), and propofol (50 mg). Tracheal intubation was successfully completed with a video laryngoscope, and norepinephrine (8 μg) was administered intravenously to manage a transient drop in BP. ABP stabilized at 140–160/75–93 mmHg, HR at 90–100 bpm, and post-intubation SpO_2 was 85%.

Anesthesia was maintained using sevoflurane (0.5–1%) and propofol (100–300 mg/h) infusion. Local infiltration anesthesia was used at the incision site, and remifentanil infusion (50–200 μg/h) began post-delivery. Hydromorphone (1.8 mg), midazolam (5 mg), and rocuronium (100 mg) were also administered. Ventilation settings included a tidal volume of 6-8 mL/kg, RR of 12/min, PEEP of 5 cmH_2O, and FiO_2 of 100%.

Surgery commenced 1 minute post-induction, and the neonate was delivered 4 minutes later. Oxytocin (10 IU) and carbetocin (100 μg) were administered to promote uterine contraction. Despite adjustments to PEEP (up to 15 cmH_2O), SpO_2 remained 80–83% with peak airway pressures of 30–35 cmH_2O. Pulmonary edema was suspected, and treatment included 20 g of albumin and 25 mg of furosemide.

2.3. Surgical and anesthetic outcomes

The procedure lasted 45 minutes, with 122 minutes of general anesthesia. Intraoperative fluid input was 700 mL (600 mL crystalloids, 100 mL albumin), blood loss was 400 mL, and urine output was 400 mL. Intraoperative ABP ranged from 120–160/70–80 mmHg, CO was 6.6–9.1 L/min, and SVV was 4–8%. The results of the patient's intraoperative arterial blood gas analysis are detailed in **Table 1**.

Table 1. Patient's intraoperative blood gas analysis

Parameter	30 minutes post-induction	60 minutes post-induction	100 minutes post-induction
pH	7.368	7.381	7.425
PO_2 (mmHg)	52	57	68
PCO_2 (mmHg)	33.1	33.1	30.8
BE (mmol/L)	-6	-3	-4
K^+ (mmol/L)	4.0	4.1	3.8
Na^+ (mmol/L)	131	129	130
Hct	0.37	0.3	0.33
Lac (mmol/L)	1.42	1.64	1.29

2.4. Postoperative course

After intensive care unit (ICU) admission, the patient remained on mechanical ventilation. Chest X-rays showed bilateral pulmonary congestion on the day of surgery, which improved by postoperative day 1. The endotracheal tube was removed 24 hours postoperatively, transitioning to HFNO (50 L/min, FiO_2 50%). By postoperative day 4, chest X-rays were normal, and the patient was discharged on day 6. She received magnesium sulfate and diuretics for one day in the ICU, with stable blood pressure maintained through antihypertensive therapy after discharge. The results of arterial blood gas analysis during the patient's ICU stay are detailed in **Table 2**. Postoperative chest X-ray images can be found in **Figure 2**.

Table 2. Patient's blood gas analysis during ICU stay

Parameter	1.5 hours post-ICU admission	6 hours post-ICU admission	12 hours post-ICU admission	23 hours post-ICU admission	3 hours post-extubation
pH	7.364	7.424	7.448	7.474	7.485
PO_2 (mmHg)	74	74	84	77	107
PCO_2 (mmHg)	37.3	34.5	35.6	33.3	32.2
BE (mmol/L)	-4	-2	1	1	1
K^+ (mmol/L)	3.6	3.7	4.2	3.9	3.6
Na^+ (mmol/L)	133	133	132	132	134
Hct	0.3	0.3	0.29	0.35	0.3
FiO_2 (%)	50	60	40	35	50

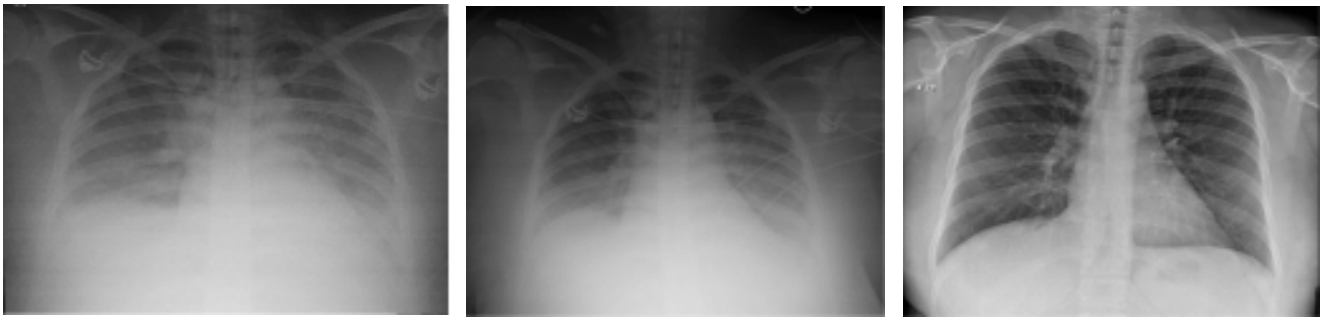

Figure 2. (Left) Chest X-ray taken on the day of surgery shows increased bilateral lung markings and enlarged hila. **(Middle)** Chest X-ray taken on the first day after surgery indicated improved bilateral pulmonary edema. **(Right)** Chest X-ray taken on the fourth day after surgery reveals normal chest X-ray findings.

2.5. Neonatal outcomes

The time from anesthesia induction to delivery was 5 minutes. The neonate required immediate intubation and had Apgar scores of 3, 7, and 8 at 1, 5, and 10 minutes, respectively. The umbilical artery pH was 7.215, and birth weight was 590 g. The neonate underwent mechanical ventilation for 32 days, transitioned to BiPAP on day 42, and achieved autonomous breathing by day 75. The neonate was discharged on day 108, weighing 2,455 g.

3. Discussion

Preeclampsia is a multisystem disorder characterized by hypertension and/or proteinuria after 20 weeks of gestation, with an incidence of approximately 3–5% in all pregnancies [1]. Its primary pathophysiological mechanism involves endothelial dysfunction, which results in widespread endothelial leakage and may lead to eclampsia, placental abruption, disseminated intravascular coagulation, severe renal failure, pulmonary edema, and hepatocellular necrosis, posing a significant risk to maternal and fetal life in severe cases [2].

Acute pulmonary edema is a leading cause of death in patients with preeclampsia and a common reason for admission to intensive care units. Its occurrence often indicates severe disease. Studies have shown that the incidence of pulmonary edema in preeclampsia is significantly higher than in normal pregnancies and correlates closely with the severity of the disease [3]. The incidence of pulmonary edema during pregnancy is approximately 0.08% [4], whereas in preeclampsia patients, it rises to 2.1%. Primiparas with preeclampsia, along with multiple pregnancies, anemia, and elevated mean arterial pressure, are more prone to developing pulmonary edema [5]. This is likely associated with systemic endothelial dysfunction, fluid retention, and increased cardiac preload and afterload caused by preeclampsia [6].

If a patient with preeclampsia experiences dyspnea and orthopnea along with signs of impaired respiratory function (e.g., tachypnea, crackles and rales on auscultation, and hypoxemia), and no alternative explanation for acute respiratory failure exists, a diagnosis of preeclampsia-related pulmonary edema should be considered. Chest X-rays and bedside ultrasound can assist in confirming the diagnosis.

For patients with severe preeclampsia, timely delivery may be the only option to ensure maternal and fetal safety [7]. When acute pulmonary edema is present, treatment depends on the severity of hypoxemia and the underlying disease. Interventions include oxygen therapy and maintaining hemodynamic stability to reduce left ventricular preload and afterload. Ventilatory support can range from nasal cannula, Venturi masks, and high-flow non-rebreather masks to HFNO, noninvasive mechanical ventilation, or endotracheal intubation with mechanical ventilation, depending on the severity of hypoxemia and the patient's respiratory effort [8]. Hemodynamic stability can be achieved with vasodilators, diuretics, and fluid restriction.

The use of albumin in patients with pulmonary edema remains controversial. Research suggests that hypoalbuminemia increases pulmonary capillary permeability, promoting the development of pulmonary edema [9]. Some studies indicate that albumin use can improve plasma colloid osmotic pressure, thereby reducing pulmonary edema and improving oxygenation [10]. A domestic expert consensus also recommends human serum albumin solution for patients with acute respiratory distress syndrome (ARDS) and hypoalbuminemia to improve oxygenation [11]. However, albumin use may carry risks such as allergic reactions, increased infection risk, and fluid overload; some studies even suggest that high doses of albumin may increase the risk of pulmonary edema [12]. Therefore, individualized evaluation is necessary to determine whether albumin should

be used. In patients with severe preeclampsia and pulmonary edema, if hypoalbuminemia is present and routine diuretic treatment alongside fluid restriction and mechanical ventilation proves ineffective, a moderate infusion of albumin can be considered to increase colloid osmotic pressure and reduce fluid leakage, while closely monitoring hemodynamic status, electrolytes, and renal function.

Preanesthetic evaluation is crucial for patients with preeclampsia and pulmonary edema. Attention should be given to the severity of hypertension and pulmonary edema, airway assessment, hemodynamic status, coagulation function, and fetal condition. Careful consideration should be given to the type and volume of preoperative fluids, and changes in the aforementioned conditions should be monitored closely. Multidisciplinary consultations involving anesthesiologists, obstetricians, and intensivists may be necessary to formulate an individualized anesthesia plan.

All types of anesthesia carry certain risks for severe preeclampsia patients undergoing cesarean section. General anesthesia is faster to administer but carries risks of failed intubation and further complications. Spinal anesthesia allows the patient to remain conscious during delivery and reduces the impact of anesthetics on the neonate. It can mitigate neuroendocrine responses, helping to lower blood pressure. The use of intrathecal opioids also reduces postoperative pain, minimizing the risks of intubation and ventilation failure, aspiration, and blood pressure spikes or increased intracranial pressure during laryngoscopy. Thus, many studies support spinal anesthesia as the preferred method for cesarean sections in preeclampsia patients [13,14]. Spinal anesthesia is suitable for patients who are conscious and have normal coagulation function and platelet levels based on laboratory results. General anesthesia is typically used when spinal anesthesia is contraindicated, fails, or in emergencies. In such cases, individualized anesthesia management should be implemented, with preparations for difficult intubation. During anesthesia, invasive monitoring may be required in addition to standard vital signs monitoring, such as invasive arterial pressure and central venous pressure measurements.

Case reports on the anesthesia management of cesarean sections in patients with preeclampsia and pulmonary edema are limited. Alves *et al.* reported a case of preeclampsia with pulmonary edema and type 1 respiratory failure requiring oxygen therapy with a Venturi mask. The cesarean section was successfully performed under epidural anesthesia [15]. Ethy Ahammedunni *et al.* reported a case of preeclampsia with pulmonary edema and morbid obesity, in which spinal anesthesia was successfully administered in a sitting position to complete the cesarean section [16].

In the present case, the patient was diagnosed with severe preeclampsia complicated by acute pulmonary edema. Due to high suspicion of pulmonary embolism at admission, therapeutic doses of low-molecular-weight heparin were administered. As the interval between heparin administration and cesarean section was less than 24 hours, general anesthesia was chosen. Anesthesia induction was performed using titrated doses of fast-acting sedatives, analgesics, and muscle relaxants. When hypotension occurred, a single intravenous dose of low-dose norepinephrine was administered to stabilize blood pressure while achieving sufficient anesthetic depth, allowing for successful tracheal intubation. Following intubation and mechanical ventilation, the patient developed severe hypoxemia, likely due to the physiological effects of positive pressure ventilation, reduced functional residual capacity in the supine position, and exacerbation of pulmonary edema caused by postpartum uterotonic agents, increased venous return, blood loss, reduced plasma colloid osmotic pressure, and increased capillary permeability. Symptomatic treatment during anesthesia, including strict fluid restriction, diuretics, and albumin infusion, helped reduce venous return, increase colloid osmotic pressure, and enhance diuresis, ultimately improving pulmonary edema and oxygenation.

4. Conclusion

Acute pulmonary edema is a leading cause of mortality in patients with preeclampsia. For patients with severe preeclampsia complicated by pulmonary edema requiring cesarean section, both pulmonary edema and hypertension should be managed before surgery begins. When no contraindications exist, spinal anesthesia is the preferred anesthetic method for cesarean section in patients with preeclampsia. However, if general anesthesia is necessary due to contraindications for spinal anesthesia, attention should be given to the impact of general anesthesia on the patient's already compromised respiratory function, ensuring hemodynamic stability and adequate oxygenation.

Disclosure statement

The authors declare no conflict of interest.

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