Case Report

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Anesthesia Perioperative Management of a Pregnant, Severe Preeclampsia Patient with Massive Pulmonary Edema, A Case Report

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ABSTRACT

Pulmonary edema refers to accumulation of an excessive fluid in the pulmonary interstitial and alveolar spaces. It is one of the foremost serious complications of preeclampsia that should be early diagnosed in case of severe dyspnea, respiratory distress in a pregnant woman, especially in presence of preeclampsia. It is one of the most important indications of urgent pregnancy-termination. Many theories have been discussed for an explanation for this phenomenon, such as hypervolaemia, left ventricular failure and pulmonary capillary leakage, but it is still not well understood. The prognosis usually is good after adequate management and symptoms completely disappear within a few days post-delivery. In this article, we will discuss a case report of 37 years old, African pregnant female, came to emergency room in a serious condition, with severe pulmonary edema as a complication of uncontrolled preeclampsia. She was complaining of severe dyspnea, orthopnea, disturbed conscious level, diaphoretic, not maintaining saturation and extremely high BP.She was admitted to ICU for 3 hours then taken to operating theatre for termination of pregnancy through doing emergency lower segment cesarean section. We will discuss the pre-operative preparation of the patient in ICU, then the intraoperative management of the patient, and post operative prognosis of the patient.

Keywords: Preeclampsia; Pulmonary Edema; Perioperative; Spinal Anesthesia; Non-Invasive Ventilation; Anesthesia

Abbreviations: s-Eng: Soluble Endoglin; VEGF: Vascular Endothelial Growth Factor; PLGF: Placental Growth Factor; IUGR: Intrauterine Fetal Growth Restriction

Introduction

Hypertensive disorders are the most common medical complication of pregnancy, and preeclampsia has an incidence of around 3-10 % of all pregnancies in worldwide [1]. Worldwide, preeclampsia is one of the most important causes of perinatal morbidities and mortalities [2]. In the study of Sibai, et al. [3], Pulmonary edema incidience around 0.05% of the low-risk pregnancies but it may develop in up to 2.9% of pregnancies complicated by preeclampsia. Acute pulmonary oedema in pregnancy is a life-threatening condition due to multifactorial. One of the factors is the superimposed physiological changes of pregnancy and the presence of the fetus, as well as the poorly understood pathophysiology of pre-eclampsia which is associated with significant morbidity and mortality for mother and fetus. The persisting hypoxemia is a strong indication of intensive care unit admission. Death has been reported in around 10% of cases often reflecting multiorgan failure. Decision of emergency termination of pregnancy is the safest option for the patient & her baby when there is evidence of pulmonary oedema irrespective of gestational age [4].

Case Report

Case of 37 years old patient, brought by ambulance to Emergency rooms, she came with severe cough, shortness of breath, disturbed sensorium. On triage, it was found that blood pressure is extremely high, it was initially 190/120 then second reading was 220/126, saturation was low on room air 70-80%, heart rate was very high 140-150/m, respiratory rate was extremely high 55-60 breath per minute. Immediately she was admitted to Emergency resuscitation room, non-rebreathing mask was connected on 15L/m, internal medicine was called, anesthesiologist covering intensive care was called. By history from relative, the obstetrician got the clinical history, she is pregnant, and she is complaining for one week of coughing, shortness of breath, high blood pressure and she didn't

seek any medical advice due to financial reason. Ultrasound abdomen was quickly done by obstetrician in emergency room, it revealed that she is 29 weeks pregnant, and fetus was viable. Urinary catheter was inserted, Urine dipstick protein was +3, laboratory tests were sent of CBC, coagulation, serum troponin, serum pro BNP, urea, electrolytes, liver function tests, serum creatinine (Table 1).

Table 1: Laboratory results.

Component & Reference range	On arrival	2nd day	3rd day	4th day	5th day	6th day	7th day
Creatinine 0.5 - 0.9 mg/dL	1.3	1.2	1.2	0.6	0.6	0.6	0.6
Sodium 136 - 145 mmol/L	138	133	137			135	139
Potassium 3.3 - 4.8 mmol/L	4.7	4.3	3.7			3.4	3.3
Bicarbonate 20 - 28 mmol/L	12.7	15.6	18.3			22.5	23.4
Urea 12 - 40 mg/dL	52	63	30			13	23
NT-pro BNP <125 pg/mL	34.948				4.851		
Troponin <14 ng/L	117				14		
Bilirubin Total 0-1.2 mg/dL	0.4			0.2			0.1
Alkaline phosphatase 35-104 U/L	145(H)			94			101
Albumin 3.4-4.8 g/dL	2.9(L)			2.7(L)			3
C reactive protein <0.5 mg/L	45.2(H)						
Procalcitonin <0.05 ng/mL	13.3(H)						
Prothrombin time 11.5-14.5 sec	14.7(H)			14.5			
INR 0.8- 1.2	1.15			1.12			
APTT 28.6-38.2 sec	43.8(H)			37.3			
Fibrinogen. 190-430 mg/dL	482(H)						
D-dimer <05ug/mL FEU	5.37			4.1			
Mg 1.6-2.6 mg/dL		3.14(H)					

Venous blood gas was taken (Table 2: 1st result). Hydralazine 10 mg intravenously and Magnesium Salfate (MgSO4) loading dose started. On arrival of anesthesiologist, he found that patient is so much agitated, there was pallor and bilateral pitting pedal edema, dry mucus membranes, distressed, always removing the oxygen mask, sitting in bed, drowsy, diaphoretic and bilateral severe degree of lung crackles in all lung zones. Vital signs were BP 200/120, RR 55/m Saturation on oxygen 90% and HR 155/m. GCS was 11/15 [eye opening to verbal (3), verbal inappropriate words response (3), and the motor response was purposeful to painful stimulation (5)]. One dose of furosemide 40 mg was given IV in Emergency department, arrangement to transfer the patient to ICU was started. Labetalol 40 mg was given IV to control the extremely high BP.

Table 2: Blood gas results.

Component & Reference Range	1st Result Venous	2nd Result Venous	3rd Result Arterial	4th Result Arterial	5th Result Arterial	6th Result Arterial	7th Result Arterial
PH 7.35 - 7.45	7.161 (L)	7.101(L)	7.198	7.349	7.411	7.360	7.364
PCO2							
35 - 45 mmHg	38.7	41.2	26.0 (L)	23.7 (L)	24.8 (L)	27.3 (L)	29.6 (L)
PO2 83 - 108 mmHg	24.2 (L)	25.2	244 (H)	263 (H)	128 (H)	148 (H)	232 (H)
HCO_3 (P) 21 - 28 mmol/L	12.7 (L)	11.4 (L)	11.9 (L)	15.2 (L)	17.9 (L)	17.2 (L)	18.5 (L)
CTHB 11.0 - 15.0 g/dL	9.2(L)	9.6(L)	8.9(L)	6.6(L)	7.1 (L)	8.7 (L)	11.4
SO_2 95 - 99 %	21.4 (L)	21.3	100	100	99.7 (H)	100.0 (H)	100.0 (H)
FO2HB 95 - 98 %	21(L)	21.0	98.7	98.9	98.1 (H)	97.8	98.6 (H)
FCOHB 0.5 - 1.5 %	0.8	0.6	1.2	1.5	1.5	1.8 (H)	1.0
FMETHB 0.5 - 1.5 %	1.1	0.8	0.2	0.3	0.1 (L)	0.7	0.5
CTBIL mg/dL	0.6	0.5	0.3	0.2	0.0	0.1	0.2
FHBF %	1.1	NO VALUE	NO Value	NO Value	NO VALUE	NO VALUE	NO VALUE
K 3.4 - 5.0 mmol/L	4.7	4.7	4.7	4.8	4.9	5.3 (H)	4.4
NA 134 - 143 mmol/L	141	144	138	137	137	137	138
Ionised Calcium 1.15 - 1.29 mmol/L	1.14 (L)	1.15	1.15	0.94	1.06 (L)	1.05 (L)	1.16
CL 97 - 108 mmol/L	113 (H)	113	112	115	112 (H)	112 (H)	112 (H)
Glucose 60 - 100 mg/dL	62	49	47	249	152 (H)	163 (H)	129 (H)
Lactic Acid 0.5 - 1.6 mmol/L	6.7 (H)	7.3(H)	6.8(H)	3.7	2.9 (H)	2.9 (H)	1.4
BASE (ECF) (-) mmol/L	14.8	16.8	18.0	12.6	8.8	10.0	8.5

She arrived to ICU, with same condition, monitor was connected, all intubation equipment is kept ready, patient was positioned head up position 30 o. Anesthesiologist in ICU started a trial of non-invasive CPAP with settings of FIO2 100%, PS 15, PEEP 8, face mask of CPAP was secured, initial Venous blood gas was sent (Table 2: 2nd result). Bp was very high and lung crackles was still audible, so labetalol 80 mg bolus was given, and labetalol infusion 1 mg/min was started, second dose of furosemide 40 mg was given intravenously With assistance of ultrasound, and under aseptic precautions, wide bore canula 16 G was inserted in left external jugular vein and 20G arterial canula were inserted. Initial blood gas was sent. (Table 2 - 3rd result). The result showed metabolic Acidosis, with hypoglycemia and high lactate. Urinary catheter was inserted, there was no urine. 200 ml of D10 + 0.9% Normal saline was started over on hour with careful monitoring of clinical feature of worsening of pulmonary edema. Continuous CTG was started.Bedside lung ultrasound was done by anesthesiologist covering ICU, it showed multiple B-lines. ECG was done it showed non-specific ST segment and T wave changes (Figure 1) Laboratory results came (Tables 1 & 2: on admission result), it showed high creatinine, high alkaline phosphatase, extremely high pro BNP, high troponin, high septic markers (procalcitonin, CRP) and CBC showed low hemoglobin, high WBCs. After starting CPAP, patient started to be quiet, Saturation started to improve to 98-100%, chest Xray was done after an hour of starting CPAP (Figure 2) it shows: Bilateral central hilar and para-hilar patchy areas of heterogenous opacities associated with intervening areas of air bronchograms favor pulmonary edema Dominant on the right lower segment mass like opacity. Congested both lungs and prominent vascular markings

seen. Cardiac shadow hardly commented upon cardiothoracic ratio 0.58. Patent both Costophrenic angels.

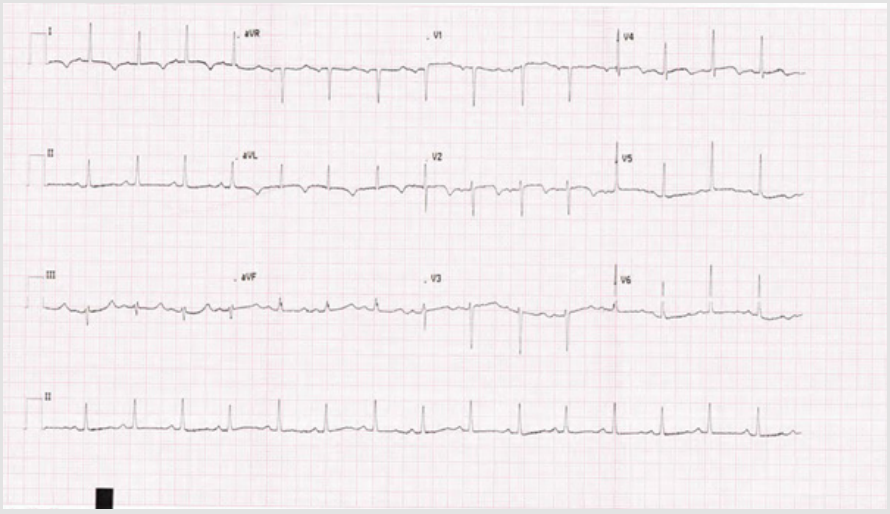

Figure 1: ECG.

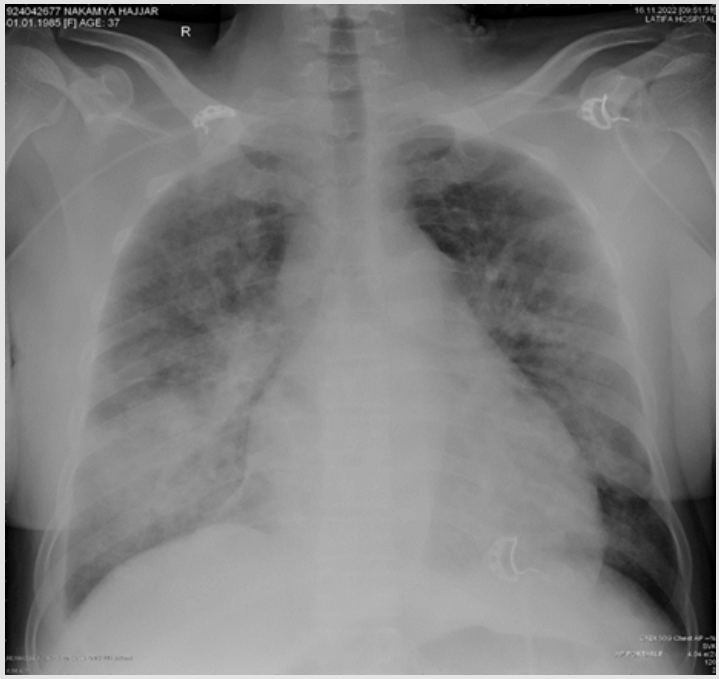

Figure 2: Initial Xray.

ABG was repeated after an hour of initiation of CPAP non-invasive ventilation (Table 2: 4th result), it showed improvement of PH, lactic Acid, serum bicarbonate and serum glucose, but low hemoglobin so, Cross matching was sent in view of low HB. Second assessment of the patient and situation was done after one and half hour of admission, it shows improvement of vital signs, BP became 155/100, heart rate 110/m RR became 28/m saturation became 98% but urine output was very low 10 ml/hr, second very limited bolus of fluid was started 150 ml over one hour. CPAP setting was revised to FIO2 70% PS 14 PEEP kept 8, ABG was sent after an hour of CPAP changes (Table 2: 5th result), it shows further improvement of PH, lactic acid. After finishing the bolus, urine output started to increase, initially it was 150 ml, IV fluid rate re-adjusted to 70 ml/hr (1 ml/kg/hr), GCS showed improvement (13/15), mild confusion was still there. Lung auscultation showed improvement and regression of crackles.

Complete blood count was repeated (Table 3: after two hours

of admission) it shows further increase in WBC, and further drop in Hemoglobin. First unit of PRBCs were requested and started over an hour. Obstetrician on-call noticed repeated variable fetal deceleration, so she decided Emergency cesarean section. Patient was transferred to OT on non-rebreathing mask 10 L/m, she was examined before taking decision of anesthesia, anesthesiologist on call took a decision of Spinal anesthesia as she can lie flat supine position using non rebreathing mask, to avoid pressor effect of intubation and Spinal anesthesia will decrease afterload of the heart and will improve pulmonary edema. 11 mg of Heavy bupivacaine 0.5% with 100 mcq preservative free morphine and 15 mcq of fentanyl was injected in L4-L5 space using Whitacre needle 26 G under complete aseptic precautions. Her Bp was stable till Baby delivery, after that she started to have hypotension. Phenylephrine boluses was used 3 times 40 mcq each dose, then nor epinephrine 0.05 mcq/kg/min was started. Under complete aseptic precautions, and after local anesthesia infiltration, Internal jugular vein central line was inserted under US guidance.

Table 3: CBC results.

Component & Reference range	On admission	After 2H of arrival	Postop result	Third day of surgery
WBC COUNT 3.6 - 11.0 10:3/ uL	11.7 (H)	16.8 (H)	15.0 (H)	15.9 (H)
RBC COUNT 3.80 - 4.80 10:6/ uL	4.87 (H)	4.17	4.76	4.39
HEMOGLOBIN, BLOOD 12.0 - 15.0 g/dL	8.2 (L)	7.0 (L)	9.4 (L)	9.0 (L)
HEMATOCRIT 36.0 - 46.0 %	29.4 (L)	24.1 (L)	31.0 (L)	28.8 (L)
MCV 77.0 - 95.0 fL	60.4 (L)	57.8 (L)	65.0 (L)	65.6 (L)
MCH 27.0 - 32.0 pg	16.9 (L)	16.9 (L)	19.8 (L)	20.5 (L)
MCHC 31.5 - 34.5 g/dL	28.0 (L)	29.2 (L)	30.5 (L)	31.2 (L)
RDW 11.5 - 14.0 %	27.1 (H)	26.8 (H)	35.3 (H)	35.0 (H)
PLATELETS COUNT 150 - 410 10:3/uL	276	196	218	180
MPV 7.4 - 10.4 fL	8.9	9.0	9.2	8.5

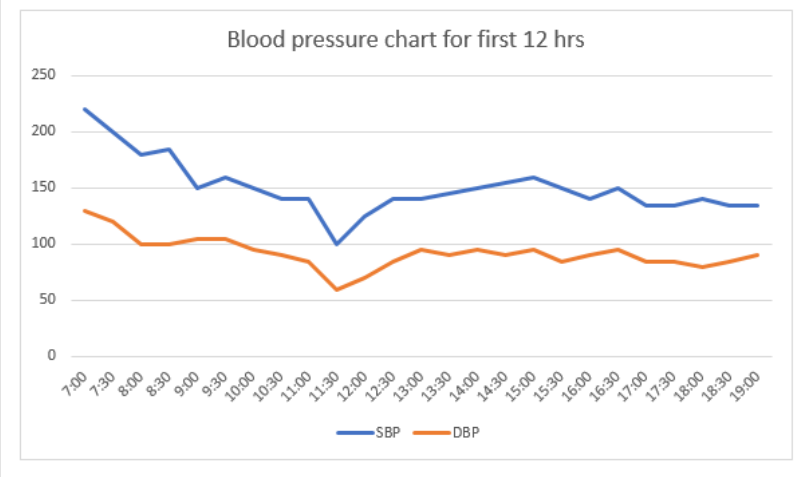

Figure 3: Trends of BP for first 12 hours.

Two units of PRBCs were requested, Blood transfusion started; she received intraoperatively 2 units of PRBCs over an hour (time of procedure).Her BP started to be stabilized after giving blood, Nor-epinephrine started to be tapered and finally stopped. Surgery finished successfully, it took one hour, estimated blood loss was in range of 700 ml. Urine output during surgery was 180 ml/hr. blood gas was done at the end of procedure (Table 2: 6th result) She was transferred back to ICU, on 10 L/ non rebreathing face mask. Her BP started to rise again, so labetalol infusion 1 mg/min started again (see figure 3: BP trend in first 12 hours). MgSO4 1gm/hr continued in the ICU for 48 hours, with continuous clinical monitoring of signs of hypermagnesemia and serum Mg was sent after 24 hours of starting MGSO4. (Table 1:

Mg result) After 4 hours of her surgery, her sensorium returned to normal GCS 15/15, urine output was maintained, BP was high normal level SBP 135-140 and DBP 85-90 on labetalol infusion and MgSO4 infusion. Blood gas was done (Table 2: 7th result).

Chest Xray was repeated, to confirm central line position, and check the progress of pulmonary edema. It showed central line in place, and dramatic improvement of pulmonary edema (Figure 4) Anticoagulant started after 12 hours of surgery. Night of surgery, oxygen was weaned gradually till discontinued successfully after 24 hours of surgery. She stayed two days in ICU, for monitoring of BP, Urine output, conscious level, early signs of hypermagnesemia, she was improving gradually, IV infusions for labetalol was stopped and replaced with oral antihypertensive then she was discharged to post operative ward. She stayed 5 days in the post-operative ward for controlling BP, various laboratory tests were repeated through her stay in the hospital (Table 1), she was discharged home with controlled BP with oral medications.

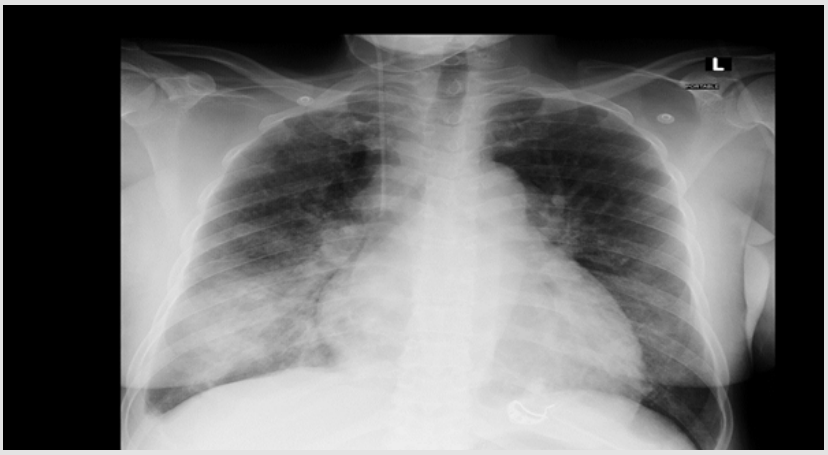

Figure 4: Post operative Xray.

Discussion

Preeclampsia is thought to be due to an abnormal development of the placenta, with a failure of proper penetration of the cytotrophoblast cells into the myometrial segment of the spiral arteries and it may cause hypoperfusion [5]. Bhorat I, et al. [6] suggested that the local hypoperfusion results in release of various substances, including inflammatory cytokines and antiangiogenic proteins, including the soluble form fms like tyrosine kinase 1 (sFlt 1) and soluble endoglin (s-Eng) as well as low circulating maternal concentrations of vascular endothelial growth factor (VEGF) and placental growth factor (PLGF) that contribute to systemic endothelial response, manifested clinically as high Blood pressure, preeclampsia and intrauterine fetal growth restriction (IUGR). The situation may be worsened with short-term cardiovascular complications of preeclampsia include heart failure, pulmonary edema, and stroke [7]. Pulmonary edema (PE) is a life-threatening condition that can be associated with preeclampsia. Incidence of PE with severe preeclampsia is 2.9% [8]. The causes of pulmonary edema in severe preeclampsia are often multifactorial. According to the Starling equation, any factor that results in a decrease in colloidal osmotic pressure (or in the colloidal osmotic pressure/pulmonary capillary wedge pressure gradient), an increase in capillary permeability, or an increase in intravascular hydrostatic pressure will lead to extravasation of fluid from the lung vasculature to lung endothelium and predispose to the development of pulmonary oedema [4].

Many mechanisms have been suggested to explain the pathology of pulmonary oedema in severe pre-eclampsia including hypervolaemia, left ventricular failure and pulmonary capillary leakage [9]. Pulmonary oedema could be due to a combination of multifactor including the fluid accumulation, retention, and failure of fluid redistribution from the systemic circulation to the pulmonary circulation due to massive venoconstriction of systemic circulation [10]. However, it is thought that rapid increase in systemic vascular resistance significantly induces changes in afterload left ventricular myocardium contributing to diastolic filling abnormalities and development of myocardial ischemia with high incidence of development of heart failure, pulmonary edema and/or sudden death [6].

Pulmonary edema in preeclampsia patient may be associated with many symptoms (shortness of breath, orthopnoea, agitation, and cough) and signs (tachycardia, tachypnoea, crackles and wheeze on lung auscultation, cardiac S3 gallop rhythm and murmurs on heart auscultation, low oxygen saturation). Typical chest X-ray picture and/or lung ultrasound show the characteristic Kerley-B lines and pulmonary infiltrates [11]. The management plan of pulmonary edema in preeclampsia is almost the same as nonpregnant patients: oxygen therapy, decrease preload through water restriction and intravenous furosemide (80 mg initially) and central hemodynamic monitoring.

Reduction in afterload is gained with the use of vasodilators (e.g.: hydralazine, nifedipine, nitroglycerine, Na nitroprusside) [12]. Non-invasive ventilation should be tried as the initial technique before tracheal intubation [13].

The routine use of magnesium sulphate MgSO4 for seizure prophylaxis in women with preeclampsia with severe features is an established obstetric practice. There is clear evidence that MgSO4 is the best available agent for prevention of recurrent seizures in women with eclampsia thus, its use has been extended to seizure prophylaxis in women with preeclampsia with severe features [14]. A meta-analysis of the available data identified six trials involving 11,444 women that compared MgSO4 for the treatment of preeclampsia with either placebo or no anticonvulsant. [15] Magnesium decreased the risk for developing eclampsia, decrease the risk for maternal death, but no effect on serious maternal morbidity. Additionally, MgSO4 therapy reduced the risk for placental abruption. It did not adversely affect fetal and/or neonatal outcomes, including stillbirth, perinatal death, or neurosensory disability. Treatment with magnesium increased the risk for maternal respiratory depression and cesarean delivery [15].

Other side effects that were significantly more common in those treated with magnesium included feeling warm or flushed, nausea/vomiting, muscle weakness, hypotension, dizziness, drowsiness/confusion, and headache. In general, magnesium sulfate is not indicated for seizure prevention in preeclampsia without severe features [16]. The mechanism of the anticonvulsant effect of magnesium is not well understood. It was previously believed that eclamptic seizures were the result of cerebral vasospasm, and it was also believed that the cerebral vasodilating properties of magnesium reduced the rate of eclamptic seizures by relieving vasospasm. [17] However, there is evidence that abrupt, sustained blood pressure elevation overwhelms myogenic vasoconstriction and causes forced dilation of the cerebral vessels, hyperperfusion, and cerebral edema [18].

Although preeclampsia is associated with intravascular volume depletion, the optimal approach to fluid management remains controversial, given potential dysfunction of the pulmonary endothelial glycocalyx and renal glomeruloendotheliosis. A 2011 systematic review [19] found insufficient evi¬dence of maternal or neonatal benefit of plasma volume expansion in preeclampsia. Given the heterogeneity of the disease, clinicians should restrict fluids in these patients unless monitoring is used (e.g., invasive blood pressure moni¬toring with or without noninvasive cardiac output monitoring, TTE) to assess response to fluid administration. Volume expansion is not recommended, and fluids should be limited to 80 mL/h or 1 mL/kg/h. In the case of hemorrhage, losses should be replaced appropriately. Administration of addi¬tional fluid may be considered before intravenous hydralazine, neuraxial anesthesia, or immediate delivery. Care must be taken not to overtreat oliguria with fluids; a fluid challenge is recommended in oliguric patients only if a volume deficit is suspected or can be confirmed [20]. The occurrence of pulmonary oedema in preeclampsia patient is one of the strong indications of urgent termination of pregnancy [12] The choice of anesthesia in severe preeclampsia is controversial, the traditional view was that spinal anesthesia is relatively contraindicated in severe preeclampsia because of the possibility of marked hypotension as a result of the rapid onset of spinal anesthesia–induced sympathetic blockade. However, this concern is not supported by evidence.

Wallace, et al. [21] randomized 80 women with severe preeclampsia who required cesarean delivery to receive general, epidural, or CSE anesthesia. There was no significant difference between the CSE and epidural anesthesia groups in maternal mean arterial pressure over time. Notably, the initial spinal dose in the CSE group (hyperbaric bupivacaine 11.25 mg) is a dose comparable to that often used for a single-shot spinal technique Aya, et al. [22] compared women who had severe preeclampsia with healthy pregnant women (both preterm and term) and found that the risk for significant hypotension (defined as requiring the administration of ephedrine) was significantly lower in the preeclampsia groups than in the healthy control groups. The authors speculated that the known increased vascular sensitivity to vasoconstrictors may explain the infrequent incidence of hypotension after spinal anesthesia and the ease with which mean arterial blood pressure can be restored to baseline with small doses of vasopressor.

In contrast, a randomized multicentre study [23] comparing the hemodynamic effects of spinal anesthesia with epidural anesthesia for cesarean delivery in women with severe preeclampsia found that significantly more women in the spinal anesthesia group experienced hypotension. However, the duration of hypotension was less than 1 minute in both groups and, although more ephedrine was used in the spinal group than in the epidural group, hypotension was easily treated in both groups. In addition, there was no significant difference in neonatal outcome between infants whose mothers received spinal anesthesia compared with those whose mothers received epidural anesthesia. Another study suggested that spinal anesthesia has little effect on cardiac output in severely preeclamptic women, and reduction in afterload was modest [24]. General anesthesia is less desirable than neuraxial anesthesia because of the possibility of difficult tracheal intubation secondary to airway edema and the transient but severe hypertension that accompanies tracheal intubation and extubation. Nonetheless, there are situations in which general anesthesia is the best anesthetic option. Clinical indications include severe ongoing maternal hemorrhage, sustained fetal bradycardia with a reassuring maternal airway examination, and contraindication of regional anesthesia e.g. coagulopathy.

Statements

Acknowledgment

None.

Statement of Ethics

Written informed consent was obtained from the patient mother for publication of this case report and any accompanying images. Ethical approval is not required according to Dubai Health Authority committee policies.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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None.

Author Contributions

Ahmed Hashey contributed to the manuscript, discussion, and conclusion.

Data Availability Statement

All data generated or analyzed during this case report are included in this article. Further enquiries can be directed to the corresponding author.

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