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CASE REPORT

Difficult Airway with HELLP Syndrome

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ABSTRACT

Pregnancy due to its generalized edematous state is known to have a difficult airway. Preeclampsia makes this difficult airway more difficult and if its coupled with HELLP syndrome which becomes anesthetic's worst nightmare.

We managed a similar case in our hospital, with multisystem involvement, for an emergency cesarean section. Surgery was carried on Proseal LMA (laryngeal mask airway) due to difficult intubation but for definitive airway we resorted to fiber-optic intubation, thus avoiding invasive airway.

Keywords: HELLP syndrome, Difficult airway, Severe pre-eclampsia.

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INTRODUCTION

HELLP (hemolysis, elevated liver enzymes and low platelets) syndrome was first defined by Weinstein in 1982.[1] The reported incidence of HELLP syndrome among patients with preeclampsia ranges from 4 to 12% depending on the criteria that are used to define the syndrome.

Diagnosis requires at least two of the following:

1. Hemolysis:
   - Abnormal peripheral smear (schistocytes, burr cells)
   - Elevated serum bilirubin (≥1.2 mg/dl)
   - Low serum haptoglobin
2. Elevated liver enzymes:
   - Aspartate aminotransferase or alanine aminotransferase at least twice the upper level of normal
   - Lactate dehydrogenase at least twice the upper level of normal. This value is also elevated in severe hemolysis
3. Low platelets:
   - <100,000/mm^3

Thus, along with liver dysfunction, there is an increased maternal and fetal morbidity and mortality due to the low platelet count as well as hemolysis.[2] In addition, these patients have an increased risk of developing consumption coagulopathy, acute renal failure, abruptio placentae, cerebral and pulmonary edema, liver hematoma and hypovolemic shock.[3]

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Maternal mortality in various studies has been reported to vary between 0 and 24% in patients with HELLP.[4] Perinatal mortality has been reported between 37 and 85%.[4] The only genuine treatment, like eclampsia, is accepted to be termination of the pregnancy. Antepartum corticosteroids are an accepted modality to enhance fetal lung maturity. It is also wise to transfer the patient into a tertiary level care so that her health can be well-monitored and the baby can be promptly shifted in an intensive care unit (ICU).

Complicated pregnancies require good understanding of maternal physiology and pathology, and careful monitoring as well as prompt action on part of an anesthesiologist for better maternal and neonatal outcome.

CASE REPORT

Here, we present a case report of one such patient dealt at our hospital.

A 41-year-old woman, third gravida, with one abortion and one live born baby in the past, came to us at 29 weeks gestation. She had delivered a live, full-term male baby 5 years back and did not have any medical complications in that pregnancy. She had a spontaneous miscarriage at 2 months amenorrhea 3 years back. Presently she was having an *in vitro* fertilization (IVF) conception, referred from a nursing home and was admitted to the ICU with complaint of acute severe pain in the epigastrium.

On general examination, patient was alert and oriented. Vitals showed a pulse rate of 80/minutes, blood pressure of 220/110 mm Hg. She also had bilateral pitting pedal edema, with mild icterus. Systemic examination revealed no obvious abnormality.

A provisional diagnosis of severe preeclampsia with 7 months of amenorrhea was made.

Blood investigations revealed the following:

Hemoglobin	6.6 gm/dl	Total bilirubin	4.1 (D-2.0/I-2, 1) mg/dl
PCV	18.5	SGOT	1348
WBC	16,500/mm^3	SGPT	642
Platelet	68,000/mm^3	Total protein	6.4 (A-2.9/G-3.5) mEq/l
INR	1.2	Na	135 mEq/l
PTT	29/28.5	K	4.8 mEq/l
FDP	178	Cl	104 mEq/l
Urine protein	3+	Serum creatinine	0.8

Chest X-ray showed bilateral prominent brochovascular markings (Fig. 1).

USG (ultrasound) abdomen showed mild areas of subcapsular hemorrhages in the liver and single intrauterine live fetus corresponding to 29 weeks of gestation.

Hence, a final diagnosis of severe preeclampsia with HELLP syndrome with 29 weeks of gestation was made.

In the ICU, two wide bore (18G) peripheral IV line were secured. Antihypertensive medications were started to control the blood pressure-alpha methyldopa 250 mg QDS, nifedipine R 20 mg BD and labetalol infusion 100 mg/50 ml. Magnesium sulfate ($MgSO_4$) infusion as per Zuspan regimen was started. A loading dose of 4 gm I/V of $MgSO_4$ was followed by an infusion of 1 gm/hour which was continued for 24 hours. Patient's urine output decreased, hence, central line attempts were made, but were unsuccessful leading to hematoma in the neck. Decision for emergency LSCS was taken considering uncontrolled hypertension and worsening maternal health.

Adequate blood, platelets and fresh frozen plasma units were cross-matched and issued. Informed and written consent were taken.

On arrival to the theater, two peripheral wide bore IV lines were secured. The labetalol infusion was continued from ICU and sodium nitroprusside (SNP) infusion was started slowly titrating the dose to achieve BP control preinduction. Platelet concentrates were started at induction itself.

Among the monitors, ECG, SpO_2, invasive blood pressure monitor and bispectral monitor was applied. A vigilant watch on the patient also made sure any signs of impending eclamptic fit were keenly observed.

Patient was preoxygenated with 100% O_2 for 5 minutes. Rapid sequence induction with cricoids pressure done with measured doses of propofol, rocuronium 5 mg (for pre-curarization) and succinyl choline 75 mg was given for intubation. Direct laryngoscopy with Macintosh blade number 3 showed gross supralaryngeal edema with no visualization of the cords (Fig. 2).

Patient was ventilated and second attempt of laryngoscopy was performed which also failed to show larynx. In view of coagulopathy to avoid injury and bleeding Proseal LMA (laryngeal mask airway) number 3 with Ryle's tube was passed. Patient could be ventilated with LMA with satisfactory capnography and was put on volume controlled ventilation with tidal volume of 450 ml and respiratory rate of 12/minutes.

Patient was maintained on oxygen/air with 60% FiO_2. Inhalational agents were avoided to prevent uterine hypotony. Propofol infusion started and titrated to BIS score, along with atracurium bolus as a muscle relaxant. Midazolam 2 mg was given. Blood pressure was maintained around 130/80 mm Hg. Baby was delivered, shifted to NICU (neonatal intensive care unit) and eventually did well. Fentanyl 100 µg was given after delivery of baby.

Uterus showed delayed contraction, which was managed by oxytocin and methyl-ergometrine 5 IU and 0.2 mg respectively intramuscularly. Carboprost tromethamine IM 250 µg keeping in mind possibility of intramuscular hematoma and tranexamic acid 500 mg IV was given after the delivery of baby.

The total intake for the duration of 100 minutes was 8 units of platelets, 4 FFP (fresh frozen plasma units), three packed cell units with urine output being 30 ml/hour. Blood loss estimated was approximately 1,500 ml. With decreased urine output, injection of Frusemide 60 mg and 100 ml mannitol was given. Patient developed bilateral basal crepitations, toward the end of the surgery, hence, the decision of postoperative ventilation was taken which made definitive airway mandatory.

Attempt of fibreoptic bronchoscopy was considered to avoid tracheostomy keeping an ENT surgeon standby. Oral fiber-optic bronchoscopy (FOB) showed severe edematous epiglottis and supraglottic edema completely occluding the larynx. Scope was gently passed through the soft supraglottic tissue and cords were indentified. Cuffed endotracheal tube no. 7 was passed and patient continued on controlled mode ventilation.

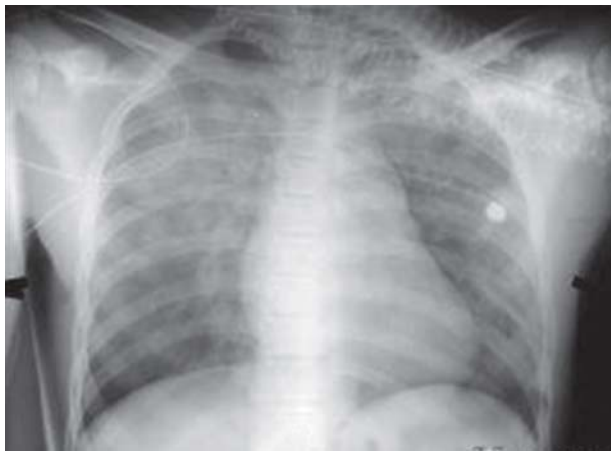

Fig. 1: X-ray showing bilateral bronchovascular markings

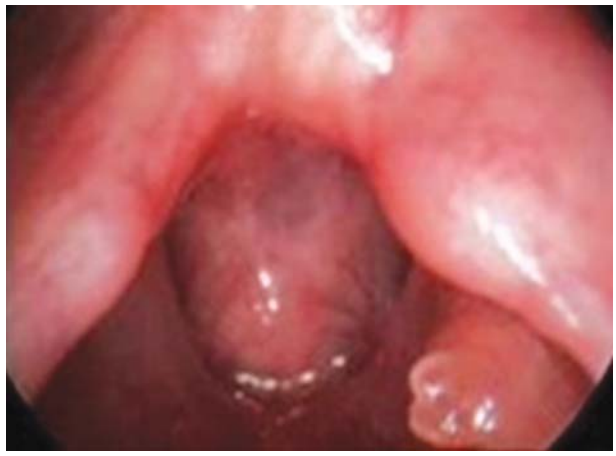

Fig. 2: Laryngoscopy showing supralaryngeal edema

Patient was not reversed and shifted to ICU with AMBU ventilation and connected to the ventilator and kept on control mode ventilation. Repeat blood investigations showed an increasing platelet count and hemoglobin. Frusemide infusion started to increase the urine output which normalized on the second postoperative day with serum creatinine raising to maximum of 5.3 mg/dl and then slowly declining. Sodium nitroprusside and labetalol infusion tapered off by postoperative day (POD) 2 and patient was kept on nifedipine orally. Pulmonary edema settled with subsequent arterial blood gas parameters improving. Patient was gradually weaned off the ventilator and extubated on POD 4. After the vitals stabilized patient who was shifted to the ward and was discharged after 10 days.

DISCUSSION

HELLP syndrome is a life-threatening obstetric complication considered to be a variant of preeclampsia. Both conditions usually occur during the later stages of pregnancy, or sometimes after childbirth.

Maternal mortality in treated HELLP syndrome is about 1% but it can increase with increasing complications, e.g. acute renal failure, convulsions, pulmonary edema.[5,7] As patient went in acute renal failure and pulmonary edema toward the end, postoperative ventilation became inevitable.

After inducing the patient as we failed to identify the larynx. We chose not to do multiple attempts to avoid trauma, which can be disastrous to patient with coagulopathy and increases aspiration risk. Proseal LMA provides an alternative to endotracheal tube for securing a difficult airway and was proven to be helpful in our case.[8]

In our patient, we faced many challenges namely coagulopathy, uncontrolled hypertension, difficult airway, uterine hypotonia, acute renal failure and pulmonary edema. To control blood pressure preoperatively we chose sodium nitroprusside over nitroglycerine as we did not want any further uterine relaxation which nitroglycerine could have caused.[9] We chose to maintain anesthesia by propofol infusion and not by inhalational agents as they are also potent uterine relaxants and would have contributed to uterine hypotonia and excessive hemorrhage.

On account of decreased urine output, coagulopathy, tachycardia and low hemoglobin, we chose to transfuse platelets (8 units) and fresh frozen plasma (4 units) and packed cells (3 units) rather than filling with crystalloids which would cause further dilution coagulopathy and rapid passage into the extravascular space.

Edema in preeclampsia usually gets corrected after child delivery so we chose to attempt fiber-optic intubation rather than directly resorting to a surgical airway, for if we could secure the airway, a difficult tracheostomy could be completely avoided.

CONCLUSION

We conclude that blood and blood products are better substitute when there is a preexisting coagulopathy as they give volume expansion without dilution of coagulation factors.

Multiple attempts of intubation should be avoided in a difficult airway situation.

And LMA can be a very useful tool in securing a difficult airway.

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