Fluid Overload Management in HELLP Syndrome with Pulmonary Edema Underwent Caesarean Section

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1. Introduction

The HELLP syndrome is a serious pregnancy-related syndrome characterised by the presence of haemolysis, elevated liver enzymes, and low platelets count in combination with preeclampsia/eclampsia.¹ It occurs in about 0.5-0.9% of all pregnancies and in 10-20% of cases with severe preeclampsia.²,³ Women with HELLP syndrome have a significantly increased risk of fluid overloads, such as renal failure and pulmonary oedema.⁴,⁵

Fluid overload leads to endothelial dysfunction due to inflammation and ischemia–reperfusion injury, causing damage to glycocalyx and capillary leakage. Capillary leakage leads to interstitial oedema, and due to significant loss of volume to the interstitial compartment, there is a reduction in circulating intravascular volume. All these phenomena make these patients very good candidates for rapid and severe fluid overload states leading to cardiopulmonary complications such as congestive heart failure, pulmonary oedema requiring mechanical ventilation, pulmonary restrictive defects, and reduced pulmonary compliance.⁶

2. Case Presentation

A 26-27 weeks, 40 years old parturient (body mass index of 30.48 kg/m²), third pregnancy and no history of abortion presents with fever for 3 days, nausea, no
appetite. No specific fever pattern. No significant medical history. No problem was found on the antenatal examination. Upon admission, her blood pressure was 150/90 mmHg with a heart rate of 90 bpm and respiration rate of 22 times per minute. Her peripheral oxygen saturation was 98% on room air. Her lab results were remarkable on leucocyte 12,200, thrombocyte 59,000, liver enzyme OT/PT 316/106, albumin level 2.7, NS-1 negative. She was initially diagnosed with dengue fever and was given clonidine for high blood pressure, ceftriaxone for leucocytopenia and ringer lactate solution 2,000 mL/24 hours.

On the second day, she experienced dyspnoea, sweating, with cold extremities. Her blood pressure rose to 170/120 mmHg, heart rate 103 bpm, and respiration 30 times/min, with 97% SpO2 on room air. Other physical findings were unremarkable. Upon workup diagnostic tests, we found elevated aspartate transaminase (AST, 316 IU/L) and alanine transaminase (ALT, 166 IU/L). We also found proteinuria. She was then given hydroxyethyl starch (HES) solution 1,000 mL/24 hours, Ringer’s acetate 500 mL/24 hours, Adalat 5 mg sublingual, and MgSO4 40% 10 grams IV.

On the third day, she reported worsening breathlessness, with the presence of frothy sputum upon coughing. Her blood pressure rose to 190/120 mmHg, heart rate of 136 bpm, respiration 40 times/min, with SpO2 of 55% on room air. Blood gas analysis returned acidosis (pH 7.347, pO2: 45.7 mmHg, pCO2 43 mmHg, HCO3: 25.3 mmol/L, SaO2: 74.4%). Emergency obstetric consultation resulted in a diagnosis of HELLP syndrome with pulmonary oedema. She was then prescribed additional sublingual nifedipine 5 mg to lower the blood pressure. She then consented to an emergent caesarean section under general anaesthesia.

Additional blood sample was collected for further workup studies. Apart from standard ASA monitoring, we also applied a central line to the patient. A new course of antibiotics (Meropenem 1 g every 8 hours) was started, along with dobutamine (continuous infusion, 5 mcg/kg/min) and norepinephrine (0.1 mcg/kg/min). Fluid intake 500 cc; blood loss 200 cc, fluid balance + 300 cc. Post operation the patient was not extubated and then transferred to ICU for further monitoring. After the operation, laboratory result were obtained, lactate: 3.3 then resuscitation with 5% albumin was performed.

The first day in ICU, the patient is on ventilator’s mode SIMV 12 PEEP 12 tidal volume 400 ml oxygen fraction 100%. Blood gas analysis result: pH: 7.272, pO₂: 54.6, pCO₂: 152, HCO₃⁻: 25.3, BE: -1.3, SpO₂: 99%. We also calculate the first-day fluid balance. Fluid input: 1786 cc, Fluid Output 3400 cc. Urine output 2.0 cc / kg of bodyweight / hour. Fluid balance: -1614 cc. Fluid overload: <10%. CRX pneumonia DD/pulmonary edema. Echo EF 12-18%, IVC dilated over volume impression, CVP 23 mmHg. Cardiac output: 2.5. Therapy changes: furosemide 5-10 mg/hour, nicardipine 2 mg/ hour and morphine 2 mg/hour.


The third day in ICU; rhonchi diminished and began to use T-pieces. Blood gas analysis result: pH: 7.464, pO₂: 41.4, pCO₂: 160.9, HCO₃⁻: 29.8, BE: 6.2, SpO₂: 99.5% Fluid balance: fluid input 2185 cc, fluid output 5330 cc, 3 cc / hour of urine output, Fluid balance: -3145 cc / 24 hours. Cumulative balance - 5404 cc. Fluid overload: <10%. Furosemide therapy 17 mg / hour. Leukocytes 13600, pro BNP 425, lactate 1.3. Echo of 55% cardiac output 3.5 CVP 11 mmHg. 2 hours of t-piece and clinical improvement then extubation was performed. The patient was transferred to the ward then. The patient was discharged on the 5th day after surgery in good condition.
Table 1. Follow-up patient in ICU.

<table>
<thead>
<tr>
<th></th>
<th>1st Day</th>
<th>2nd day</th>
<th>3rd day</th>
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<tbody>
<tr>
<td>Blood gas analysis</td>
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<tr>
<td>pH</td>
<td>7.272</td>
<td>7.385</td>
<td>7.464</td>
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<tr>
<td>pO₂</td>
<td>54.6</td>
<td>48.6</td>
<td>41.4</td>
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<tr>
<td>pCO₂</td>
<td>152</td>
<td>258</td>
<td>160.9</td>
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<tr>
<td>HCO₃</td>
<td>25.3</td>
<td>29.4</td>
<td>29.8</td>
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<tr>
<td>BE</td>
<td>-1.3</td>
<td>4.2</td>
<td>6.2</td>
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<tr>
<td>SpO₂</td>
<td>99</td>
<td>99.8</td>
<td>99.5</td>
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<tr>
<td>Balance</td>
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<tr>
<td>Input</td>
<td>1786 ml</td>
<td>2117 ml</td>
<td>2185 ml</td>
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<tr>
<td>Output</td>
<td>3400 ml</td>
<td>4430 ml</td>
<td>5330 ml</td>
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<tr>
<td>Urine output</td>
<td>2.0</td>
<td>2.0</td>
<td>3.0</td>
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<tr>
<td>Fluid balance</td>
<td>-1614 ml</td>
<td>-2259 ml</td>
<td>-3145 ml</td>
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<tr>
<td></td>
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<td>BC: -3873 ml</td>
<td>BC: -5404 ml</td>
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<tr>
<td>CRX</td>
<td>Pneumonia DD/ pulmonary edema</td>
<td>Improvements</td>
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<td></td>
<td>Echo EF 12-18%, IVC dilated over volume impression, CVP 23 mmHg. Cardiac output: 2.5</td>
<td>NT-Pro BNP 4439, lactate 1.9</td>
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<td>Treatment</td>
<td>Furosemide 5-10 mg/hour, nicardipine 2 mg/hour and morphine 2 mg/hour</td>
<td>Furosemide 10-15 mg / hour</td>
<td>Furosemide therapy 17 mg/hour</td>
</tr>
</tbody>
</table>

3. Discussion

In this report, a pregnant woman 40 years old with gestational age of 26-27 weeks (G3P2A0) presented fever for 3 days and thrombocyte 59.000. The patient was misdiagnosed as DHF and underwent fluid therapy as in the treatment of DHF. After experiencing a worsening of the condition, the patient becomes increasingly short of breath, oxygen saturation decreases, blood pressure rises. Patient was diagnosed as HELLP syndrome with pulmonary edema and sepsis. Then the obstetrician decided to terminate the pregnancy by caesarean section. Emergent caesarean section was performed with general anesthesia. The patient was intubated with low tidal volume and high peep because the patient had hypoxia and ARDS as evidenced by blood gas analysis result low PO₂, inserted CVC. We performed lactate examination intraoperative and the result is 3.3. It meant hypoperfusion to the cell. The patient was resuscitated by albumin 5% and transfused by 5 bags of thrombocyte. We gave albumin 5% because it can preserved glycocalyx. Albumin 5% will remain intravascular. We ensured perfusion to the tissue was adequate. After resuscitation we rechecked lactate level and the result was 1.9 where our target is below 2.

After surgery patient was transferred to ICU. In ICU the patient was given diuretic agent (furosemide). Diuretics agent should be given where there is evidence of systemic volume overload. The recommended initial dose of furosemide is 20-40 mg IV at admission. The dose should be up-titrated according to renal function, systolic blood pressure and history of chronic diuretic use. However, high doses are not recommended because they may be detrimental to renal function. Diuretic should always be administered according to kidney function. Management of fluid overload in this patient was challenging, especially in the patient with HELLP syndrome that we know initially has higher risk of pulmonary edema. On the second day, we changed the antibiotic empirically from meropenem combined with levofloxacin to meropenem combined with amikacin because in 48-72 hours leucocyte level increased and procalcitonin level increased. With increasing levels of procalcitonin indicates that the antibiotics are
inadequate. We also examined pro BNP levels to see if there was still fluid overload causing stretching in the right atrium.

Management of fluid overload in this patient in the ICU has lasted for 3 days. After the cumulative balance reached minus 5 liters and CVP 11 mmHg, cardiac output and micro parameters such as pH, base excess, and lactate levels were improved. There were significant hemodynamic changes and improvements in the patient’s condition as evidenced by CXR and oxygen saturation. After 3 days in ICU, the patient was carried out an awake extubation procedure. The patient was transferred to the ward then. The patient was discharged on the 5th day after surgery in good condition.

4. Conclusion

The patient showed improvement as evidenced by chest X-ray and oxygen saturation. Management of fluid overload in this patient with HELLP syndrome was challenging. On the second day in ICU, the antibiotic was changed from meropenem combined with levofloxacin to meropenem combined with amikacin because leucocyte level increased and procalcitonin level increased in 48-72 hours.

5. References