Case Report

The Unique Presentation of Massive Ascites Complicating Severe Preeclampsia and HELLP syndrome

Asites Masif sebagai Manifestasi Unik dari Preeklampsia Berat dan Sindroma HELLP

Jimmy P. Wirawan, Damar Prasmusinto
Division of Fetomaternal
Department of Obstetrics and Gynecology
Medical Faculty of Indonesia University/Dr. Cipto Mangunkusumo Hospital
Jakarta

INTRODUCTION

Preeclampsia is still a major cause of morbidity and mortality in the world, including Indonesia. Prevalence of preeclampsia and complications surrounding it estimated to be as high as 20%. Massive ascites is a rare complication of severe preeclampsia. There are only few reported cases of patients with massive ascites following preeclamptic condition. Herein we reported a patient with massive ascites in the presence of severe preeclampsia and HELLP syndrome.

CASE REPORT

A 29 years old woman, gravid 1, referred from midwife at 35 weeks of gestation due to hypertension and no fetal movement. Her antenatal history was uneventful. She admitted having leg edema and sensation of "bulging" abdomen since seven months of pregnancy. Complaints of headache and shortness of breath were evident when lying down. From physical examination, the patient was conscious. Her blood pressure was 150/80 mmHg with proteinuria (+2 on dipstick), confirmed with +3 on urinalysis. Her abdomen was larger than expected for the gestational age with no abdominal tenderness. She had bilateral leg edema. Her height was 158 cm tall with a weight of 65 kg. Neither fetal heart nor fetal movement was noticed during the examination. Her cervix was closed, posterior with no effacement.

We discovered massive ascites on ultrasound examination. There was accumulation of fluid in the subabdominal space. The diagnosis of preeclampsia was confirmed by laboratory values: thrombocyte 49 000/ul, LDH 888 U/l, and IUFD. Cesarean section was conducted. As much as 1.5 litre of ascites was evident during CS. Intra-abdominal drain was placed and ascites was about 4300 ml, 3800 ml and 1200 ml on 1st, 2nd and 3rd day. Patient was discharged after five days of care. One week follow up, patient came with good condition and controlled blood pressure and clinically no evident ascites.

Abstract

Objective: To describe a rare case of massive ascites complicating severe preeclampsia and HELLP syndrome.

Method: Reporting a case experienced and taken care in our hospital.

Result: We report a case of Mrs L, 29 years old, gravid 1, admitted at 35th week gestation, with preeclampsia, HELLP syndrome (thrombocyte 49000/ul, LDH 888 U/l), and IUFD. We found ascites pre-operatively. Cesarean section was conducted. As much as 1.5 litre of ascites was evident during CS. We put intra-abdominal drain and ascites was about 4300 ml, 3800 ml and 1200 ml on 1st, 2nd and 3rd day. Patient was discharged after five days of care. One week follow up, patient came with good condition and controlled blood pressure and clinically no evident ascites.

Conclusion: Preeclampsia is one of the major causes of maternal mortality and morbidity in the world. Manifestation of preeclampsia can range from mild preeclampsia to full blown eclamptic condition. Herein we presented a case of massive ascites complicating severe preeclampsia and HELLP syndrome.


Keywords: massive ascites, preeclampsia, HELLP syndrome

Abstrak

Tujuan: Menuturkan kasus unik asites masif menyertai preeklampsia berat dan sindroma HELLP.

Metode: Melaporkan kasus yang dialami dan ditangani pada rumah sakit kami.


Kesimpulan: Preeklampsia merupakan salah satu penyebab utama mortalitas dan morbidity maternal di dunia. Tampilan preeklampsia beragam, mulai dari preeklampsia ringan hingga kondisi ekstrem, kami melaporkan kasus asites masif yang menyertai preeklampsia berat dan sindroma HELLP.


Kata kunci: asites masif, preeklampsia, sindroma HELLP

Correspondence: Jimmy P. Wirawan, Department of Obstetrics and Gynecology, Dr. Cipto Mangunkusumo, Jakarta.
Tel.: 021-31908617; 0821-24271124. Email: jimmypanji@yahoo.com
hepatic region. Liver, spleen and both kidney seemed normal. She had a singleton pregnancy, head presentation, fetal death with biometry corresponded to 35 weeks of gestation. Estimated fetal weight 2150 gr, no spalding sign sighted. Placenta located posteriorly with no signs of abruption or prominent calcification. No uterine discontinuity was discovered.

Her laboratory results were startling. Biochemical results showed thrombocytopenia (thrombocyte 4800/ul), increase in ALT and AST level, marked increase in lactate dehydrogenase level, as shown in Table 1, correlated with HELLP (hemolysis, elevated liver enzymes and low platelets) syndrome. The condition of hypoalbuminemia (2.4g/dl) and hypercoagulable state was noted in the patient. We gave magnesium sulphate infusion and nifedipine and dexamethasone for management of severe preeclampsia and HELLP syndrome. Cesarean section was chosen to be the mode of termination because patient came with severe preeclampsia, HELLP syndrome, IUFD, and not in labor. As much as 10 units of thrombocyte concentrate transfusions were given to patient pre-operatively.

Intra operative, we found massive ascites about 1.500 ml. During removal of ascites, hemodynamic stability was noted. Neither decreasing in blood pressure nor heart rate was noted. Born baby boy 1900 gr, died, without complications. We put intra abdominal drain to observe production of ascites and prevent abdominal compartment syndrome. On the 1st, 2nd, 3rd day post-operative, production of ascites was about 4300 ml, 3800 ml and 1200 ml respectively. Analysis of ascites revealed the fluid as transudative, with low protein and normal glucose concentration. Several mesothelial and red blood cells were found.

In a study of 23 patients with pregnancy induced hypertension and ascites, Cong and Wang found albumin/globulin ratio to be < 1.5 in all patients. They concluded the finding ascsites as warrant for immediate termination of pregnancy. Low protein content was thought to reduce intravascular oncotic pressure, facilitating transudation of serum to the peritoneal cavity. Additionally, injury on glomerulus due to activated tromboplastin could do induce changes in mesothelial surface, thus contributing to development of ascites.1,2

In another study, Calvin et al. evaluated 41 women with ascites found during cesarean section conducted for various indications.4 Five patients had large volume ascites with low protein content, consistent with a transudate. Measurement of portal vein diameter and Doppler analysis of flow velocity in the portal vein showed uniform results in preeclamptic patient and normotensive pregnant women. Sibai mentioned the term atypical preeclampsia, with one of the conditions is capillary leak syndrome in which preeclampsia manifested with facial edema, ascites, pulmonary edema without hypertension.5 Patients suffering from capillary leak syndrome should be evaluated for any liver, renal and hematological abnormalities.4

Our patient was preeclamptic, with low albumin level. Albumin/globulin ratio was less than 1.5 (0.8), similar to findings by Cong and Wang. She had disseminated intravascular coagulopathy with shortening of prothrombin and activated partial tromboplastin time. Her coagulopathy was thought as a result from tromboplastin activated by preeclamptic condition and by the fetal death. These conditions predispose our patient to have an ascites, proven during cesarean section.

Management of patients with massive ascites are still numerous. Some warrants active management by directly terminating the pregnancy within 24 - 48 hour.6 Others try to conserve the pregnancy as possible with close monitoring. Ascites, especially in large volumes, would cause respiratory compromise and increased incidence of heart failure. Woods et al examined 190 patients with HELLP syndrome and found that ascites in HELLP syndrome will increase incidence of adult respiratory distress syndrome up to six fold. Those patients are more susceptible in developing heart failure up to nine fold, especially in

Table 1. Laboratory results of the patient.

<table>
<thead>
<tr>
<th></th>
<th>Pre-Op</th>
<th>1st day</th>
<th>2nd day</th>
<th>3rd day</th>
<th>4th day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (g/dl)</td>
<td>16.4</td>
<td>–</td>
<td>–</td>
<td>14</td>
<td>12</td>
</tr>
<tr>
<td>Leukocyte (/ul)</td>
<td>10.900</td>
<td>–</td>
<td>–</td>
<td>18.650</td>
<td>16710</td>
</tr>
<tr>
<td>Thrombocyte (/ul)</td>
<td>49.000</td>
<td>–</td>
<td>–</td>
<td>80.000</td>
<td>134000</td>
</tr>
<tr>
<td>ALT/AST (U/l)</td>
<td>51.40</td>
<td>–</td>
<td>–</td>
<td>16/16</td>
<td>–</td>
</tr>
<tr>
<td>Albumin (gr/dl)</td>
<td>2.4</td>
<td>–</td>
<td>–</td>
<td>2.23</td>
<td>2.16</td>
</tr>
<tr>
<td>Globulin (gr/dl)</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>2.54</td>
<td>–</td>
</tr>
<tr>
<td>Alkaline phosphate</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>94</td>
</tr>
<tr>
<td>Gamma GT</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>69</td>
</tr>
<tr>
<td>LDH (U/l)</td>
<td>–</td>
<td>888</td>
<td>–</td>
<td>634</td>
<td>–</td>
</tr>
<tr>
<td>Ureum (mg/dl)</td>
<td>–</td>
<td>80</td>
<td>–</td>
<td>72</td>
<td>–</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>–</td>
<td>1.6</td>
<td>–</td>
<td>1</td>
<td>–</td>
</tr>
<tr>
<td>Prothrombin time</td>
<td>–</td>
<td>0.86 x control</td>
<td>0.79 x control</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>aPTT</td>
<td>–</td>
<td>1.13 x control</td>
<td>1.09 x control</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Ascites production (ml)</td>
<td>4500</td>
<td>3800</td>
<td>1200</td>
<td>–</td>
<td>–</td>
</tr>
</tbody>
</table>

Patient was discharged from the ward after five days of care. There was marked improvement in general condition. Patient had controlled blood pressure, good diuresis. Albumin level 2.7 g/dl after transfusion of 100 cc of 20% albumin. Complaints of difficult breathing were non-existent.

Patient returned to follow-up one week later with good condition. Blood pressure was controlled with nifedipine. No ascites was found during physical examination. Obstetrical status revealed normal involution of the uterus.

DISCUSSION

Ascites is a rare complication of preeclampsia. Incidence of ascites in preeclampsia was reported around 1 per 1,000 patients. We searched Pubmed online with keywords "ascites" and "preeclampsia". From 1955 till 2002, fifteen case reports were published with seven of them mentioned massive ascites. Normal physiologic changes of the liver in normal pregnancy does not include the production of ascites. In the presence of preeclampsia, ascites is thought as a result of several conditions such as generalized capillary permeability, as seen such in preeclamptic condition, intra portal hepatic hypertension, or low concentrations of protein with an altered albumin/globulin ratio.

In a study of 23 patients with pregnancy induced hypertension and ascites, Cong and Wang found albumin/globulin ratio to be < 1.5 in all patients. They concluded the finding ascites as warrant for immediate termination of pregnancy. Low protein content was thought to reduce intravascular oncotic pressure, facilitating transudation of serum to the peritoneal cavity. Additionally, injury on glomerulus due to activated tromboplastin could do induce changes in mesothelial surface, thus contributing to development of ascites.1,2

In another study, Calvin et al evaluated 41 women with ascites found during cesarean section conducted for various indications.4 Five patients had large volume ascites with low protein content, consistent with a transudate. Measurement of portal vein diameter and Doppler analysis of flow velocity in the portal vein showed uniform results in preeclamptic patient and normotensive pregnant women. Sibai mentioned the term atypical preeclampsia, with one of the conditions is capillary leak syndrome in which preeclampsia manifested with facial edema, ascites, pulmonary edema without hypertension.5 Patients suffering from capillary leak syndrome should be evaluated for any liver, renal and hematological abnormalities.4

Our patient was preeclamptic, with low albumin level. Albumin/globulin ratio was less than 1.5 (0.8), similar to findings by Cong and Wang.1 She had disseminated intravascular coagulopathy with shortening of prothrombin and activated partial tromboplastin time. Her coagulopathy was thought as a result from tromboplastin activated by preeclamptic condition and by the fetal death. These conditions predispose our patient to have an ascites, proven during cesarean section.

Management of patients with massive ascites are still numerous. Some warrants active management by directly terminating the pregnancy within 24 - 48 hour.6 Others try to conserve the pregnancy as possible with close monitoring. Ascites, especially in large volumes, would cause respiratory compromise and increased incidence of heart failure. Woods et al examined 190 patients with HELLP syndrome and found that ascites in HELLP syndrome will increase incidence of adult respiratory distress syndrome up to six fold. Those patients are more susceptible in developing heart failure up to nine fold, especially in
the first 24 hours post partum.\textsuperscript{7} Idea for conserving pregnancy was because ascitic condition in preeclampsia occurred around 32.4 ± 7.4 weeks.\textsuperscript{1,3}

Lee Ko et al performed repeated paracentesis on a preeclamptic patient with massive ascites who came at 27 weeks of pregnancy, managed expectantly until 31 weeks.\textsuperscript{2} Paracentesis was conducted to the complaints of abdominal distention and breathing difficulties experienced by the patient. From history taking, our patient already had the feeling of ‘bulging’ abdomen since seven months of pregnancy, assuming she could have had the ascites before the pilling up of ascites in her peritoneal cavity as such to compromise her breathing effort.

Ashmore reported a case with bilateral hydrothorax, together with massive ascites in preeclampsia.\textsuperscript{8} We found ascitic fluid as much as 1500 milliliter intraoperatively. We put intraabdominal drain and discovered production of ascites to be massive over the next three days. Foreman inserted abdominal drain and found production of ascites to be around 12 litre over three days.\textsuperscript{9}

From the clinical course, we knew that preeclampsia, low albumin level and coagulopathy all predisposed our patient to have massive ascites. After termination of pregnancy, controlling of blood pressure, condition markedly improved. From this case, we know that massive ascites is an unusual complication in preeclampsia. Ultrasonography can be an useful tool in diagnosing ascites preoperatively. Careful assessment of history taking, clinical examination and precise usage of ultrasonography are needed to ensure this condition does not go undiagnosed.

\textbf{CONCLUSION}

In clinical practice, finding of ascites in the presence of other conditions, namely preeclampsia, will increase alertness of obstetricians to search for injuries in other organs caused by preeclampsia. More intense maternal and fetal observation should be conducted to avoid mortality in both mother and the fetus.

\textbf{REFERENCES}

5. Sibai BM, Stella CL. Diagnosis and management of atypical preeclampsia-eclampsia. AJOG. 2009; 481: 1-7