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## Case Report

# Ascites in preeclampsia: A case report

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### ABSTRACT

Preeclampsia complicates approximately 2-8% of all pregnancies and is an important cause of maternal morbidity and mortality. Ascites in preeclampsia is a rare complication. We here are presenting a case of a 28-year primigravida with 34 weeks of gestation, a known case of preeclampsia, that was referred to our institute from a primary health centre in view of impending eclampsia. Massive ascites is an unusual complication of preeclampsia which may lead to maternal respiratory compromise, which calls for active termination of the pregnancy within 24–48 hours as it cannot be cured by medical treatment. Its rarity may be due to under-reporting. The incorporation into clinical practice of evaluating the amount of ascites in patients with preeclampsia might prompt the obstetrician to more intensive and more frequent maternal and fetal surveillance to avoid maternal and fetal complications.

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## 1. Introduction

Pre-eclampsia is defined as new onset hypertension and proteinuria after 20 weeks of gestation.<sup>1</sup> Release of vasoconstrictive agents, endothelial damage, hyperpermeability of the capillaries and microangiopathic hemolysis constitute the basic pathophysiology of this condition.<sup>2</sup> Preeclampsia complicates approximately 2-8% of all pregnancies and is an important cause of maternal morbidity and mortality.<sup>3</sup> Massive ascites in preeclampsia is a rare complication. It is a manifestation of the multisystem organ exaggeration of preeclampsia. The occurrence of ascites can lead to adverse maternal outcome.<sup>4</sup> The rarity of this condition may be due to under-reporting. Here we are presenting a case of preeclampsia in which massive ascites happened to be one of the findings intraoperatively.

## 2. Case Report

A 28-year primigravida with 34 weeks of gestation was referred to our institute from a primary health centre in view of impending eclampsia. Patient complained of headache since morning with no other premonitory signs. She was a known case of preeclampsia and was on tablet labetalol 100 mg twice a day since the past 10 days. The examination showed bilateral pedal oedema. The patient's pulse was 98 per minute and blood pressure was 200/120mm of mercury. The patient's respiratory rate was 18 beats per minute however chest was auscultated and found to be clear. The per abdomen examination showed a 32 weeks sized uterus with cephalic presentation. Abdominal wall oedema was seen and fluid thrill could be elicited suggestive of ascites. The fetal heart rate was found to be 136 per minute. On per vaginal examination, cervical os was closed. On urine dipstick test, urine protein was +3. All routine laboratory investigations like complete blood count, liver and renal function tests were sent and subsequently the reports were within normal limits. The

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patient's PIH profile consisting of PT (17 seconds) INR (1.42), LDH (1140IU/L), serum uric acid (6.5mg/dl) and serum albumin(3g/dl) were deranged. NST was reactive. Patient was given 12mg betamethasone for fetal lung maturity. Magnesium sulphate prophylaxis was given as per Prichardt's regimen. Injection labetalol 20mg was administered intravenously after which her blood pressure was recorded as 180/110, that is, there was no significant drop in blood pressure. The dose was subsequently repeated. The patient was taken up for an emergency lower segment caesarean section. On laparotomy, approximately 1.5 litres straw coloured ascitic fluid was seen. A 1950gm male fetus was delivered with evidence of IUGR. Magnesium sulphate was continued for 24hrs after surgery. Mother and baby were both discharged on postoperative day 10. Ultrasonography done at time of discharge showed no ascites, urine albumin was negative and blood pressure was 130/88mm of mercury. Antihypertensives consisting of oral labetalol 100mg twice as day were continued and patient was asked to follow up after 2 weeks.

### 3. Discussion

Preeclampsia may have variable clinical presentation. The presence of reported ascites in the literature varies between 5% to 20%.<sup>5</sup> Although the cause of ascites in preeclampsia is obscure, most probable explanation is generalized capillary leak due to endothelial cell dysfunction and reduced intravascular oncotic pressure.<sup>6</sup> It is this low colloid osmotic pressure that results in effusion such as ascites.<sup>7</sup> Approximately 83 cases of large volume ascites have been reported in the setting of preeclampsia.<sup>8</sup> Many cases of preeclampsia with ascites have been recorded mainly in the form of case reports.<sup>8–11</sup> In an 11-year study of 23 patients with pregnancy induced hypertension (PIH) and ascites, Cong and Wang found that the incidence of ascites was 21.6 in 1,000 in severe PIH, and that the presence of this condition warrants termination of pregnancy as it can not be cured using medical treatment.<sup>10</sup> Vaijyanth et al estimated the incidence of ascites in preeclampsia as 1/1000 in their report.<sup>12</sup> In a study conducted in 2019 by Mbonyizina C et al. out of 106 pts with severe preeclampsia 46 had ascites on ultrasonography. There was increased morbidity seen in these patients.<sup>5</sup> In many studies massive ascites was noted at the time of caesarean section.<sup>13</sup>

Exploration of the abdomen should be conducted when ascites is found intraoperatively, to rule out intraperitoneal tumors. The abdominal cavity was explored at the time of cesarean section in our patient, no demonstrable organic cause, benign or malignant, other than pre-eclampsia could be found to explain the ascites. The clinical course of this case indicated that the underlying cause of the massive ascites was preeclampsia. The amount of ascites decreased after hypertension and proteinuria resolved. After the classic clinical signs of pre-eclampsia (hypertension, proteinuria,

edema) disappeared, the amount of peritoneal fluid also normalized. Massive ascites is an unusual complication of preeclampsia. It may lead to maternal respiratory compromise, which calls for active termination of the pregnancy within 24–48 hours as it cannot be cured by medical treatment. Its rarity may be due to under-reporting.

### 4. Conclusion

Careful clinical and ultrasonographic examinations are necessary to detect massive ascites in pregnancy. The incorporation into clinical practice of evaluating the amount of ascites in patients with preeclampsia might prompt the obstetrician to more intensive and more frequent maternal and fetal surveillance to avoid maternal and fetal complications.

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None.

### 6. Conflict of Interest

None.

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