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CASE REPORT

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# A case of preeclampsia developing massive ascites after delivery

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**Abstract**

We experienced a case of preeclampsia in which massive ascites became apparent in the postpartum period. The patient had isolated proteinuria without hypertension before delivery. The infant had fatal growth restriction and neonatal distress. Massive ascites and isolated proteinuria are important symptoms for predicting the aggravation of PE.

**KEYWORDS**

endothelial dysfunction, gestational renal dysfunction, isolated proteinuria, massive ascites, preeclampsia, vascular permeability

## 1 | INTRODUCTION

Proteinuria during pregnancy is often associated with preeclampsia (PE) but may also indicate aggravated or new-onset renal disease. As recently reviewed by Bartal et al.,[1] 2% of pregnancies exhibit isolated gestational proteinuria, and progress to PE or severe PE at a rate of up to 30%. As isolated proteinuria is part of the multisystem disease of PE, it shares many risk factors with PE.[2] In particular, patients with late-onset isolated proteinuria, at 33–36 or 37 weeks and later, were found to be at an elevated risk for PE, equal to 2.44 (95% CI: 0.80–4.08)- or 8.62 (95% CI: 7.54–9.70)-fold, respectively. In the clinic, however, proteinuria in the absence of gestational hypertension often does not flag a pregnancy as high risk or commit it to robust monitoring protocols.

Ascites is sometimes observed in PE patients. Previously, it was reported that 1.9 of 1000 PE cases exhibit ascites. This rate increases to 21.6 of 1000 patients if the PE has severe features.[3] Ascites associates with poor outcomes for both mother and neonate, but the evidence is limited.[4,5] In addition to severe PE, ascites arises from independent underlying conditions such as portal hypertension, inflammatory diseases, malignancies, and diseases associated with low hypoalbuminemia. Here, we report a patient with massive ascites becoming prominent postpartum associated with late-onset preeclampsia.

## 2 | CASE REPORT

Our patient was a 26-year-old primigravid woman managed by an obstetrics practitioner at a private clinic over the course of a naturally conceived pregnancy. She had no history of hypertension or kidney disease, but proteinuria (3+ by urine dipstick test) was observed after 35 weeks'

gestation. Blood pressure was within normal limits: sBP and dBP were between 130 and 140 mmHg, and between 70 and 140 mmHg, respectively. Ultrasonography (USG) was negative for fetal growth restriction. From 36 to 40 weeks of gestation, she gained 7 kg with leg edema. The obstetrics clinic physician did not recognize her as a high-risk pregnant woman based on the later development of isolated proteinuria without hypertension and assumed her weight gain was due to excessive diet and limited exercise.

At 40 weeks' gestation, she was admitted to the obstetrics clinic with a premature rupture of the membrane. A vacuum delivery with the Kristeller maneuver was performed due to fetal distress and maternal severe hypertension (160/102 mmHg). After transvaginal delivery with vacuum evaluation, she suffered from atonic bleeding and lost 980 g of blood rapidly. She received an extracellular fluid transfusion of 3120 ml over 6 h to cover the blood loss. The fetal growth-restricted newborn weighed 2758 g, with an Apgar score of 8 and 9 at 1 and 5 min, and an umbilical pH of 7.084. The mother was diagnosed with PE for severe maternal hypertension and FGR at the time of delivery. One day after delivery, maternal blood pressure was improved with no observation of maternal abdominal symptoms. Two days after delivery, the mother exhibited increased abdominal volume, which is almost identical to the one just before delivery. USG detected massive ascites, and she was transferred to our university hospital.

Upon entering our care, maternal blood pressure fluctuated around 140/80 mmHg, with a heart rate of 106 bpm, and SpO2 of 99% (room air) without respiratory distress. USG and computed tomography (CT) confirmed massive ascites (Figure 1). Biochemical analysis revealed abnormal serum albumin (1.8 g/dl) and creatinine (0.97 mg/dl) levels. Serum AST/ALT was within normal limits and not applicable to HELLP syndrome. A spot urine sample protein-to-creatinine ratio was 1.19 g/g Cre (Table 1). sFlt-1, measured to estimate the progression of PE after delivery, was elevated (3863 pg/ml). The serum ascites albumin gradient (SAAG) was 2.0 g/dl, which suggests transudative ascites.[6]

Based on the clinical condition, maternal hypertension, and fetal growth restriction preceded by proteinuria, the admitting diagnosis was severe preeclampsia with massive ascites. Because massive ascites was considered to be due to severe preeclampsia aggravated rapidly during delivery, no active treatment for decreasing ascites was performed. Blood pressure and blood creatinine levels were recovered over a few days. The urine volume was also increased, and body weight and waist circumference were decreased significantly. The patient was discharged on the seventh day postpartum in good condition. At the three-month follow-up, urine protein had improved to the normal level and ascites was not detectable.

## 3 | DISCUSSION

Preeclampsia is a life-threatening disorder with various symptoms, which often progress in the last trimester. Appropriate delivery timing is important to prevent adverse outcomes for mother and infant. Several severe features provide key symptoms to follow (Table 2). The pathophysiology of preeclampsia is increased vascular permeability and vascular spasm caused by endothelial dysfunction. Because effusion into the body cavity in preeclampsia is generally explained by increased capillary permeability due to endothelial cell dysfunction and reduced intravascular oncotic pressure, ascites is thought to be an early event in aggravated preeclampsia.[7] Cong et al.[3] report an ascites incidence in severe preeclampsia of 21.6 of 1000 pregnancies. Vaijyanath et al.[8] estimate the incidence of ascites at 8 in 1000 patients during pregnancy, with ascites developing from 27 to 31 weeks' gestation. In

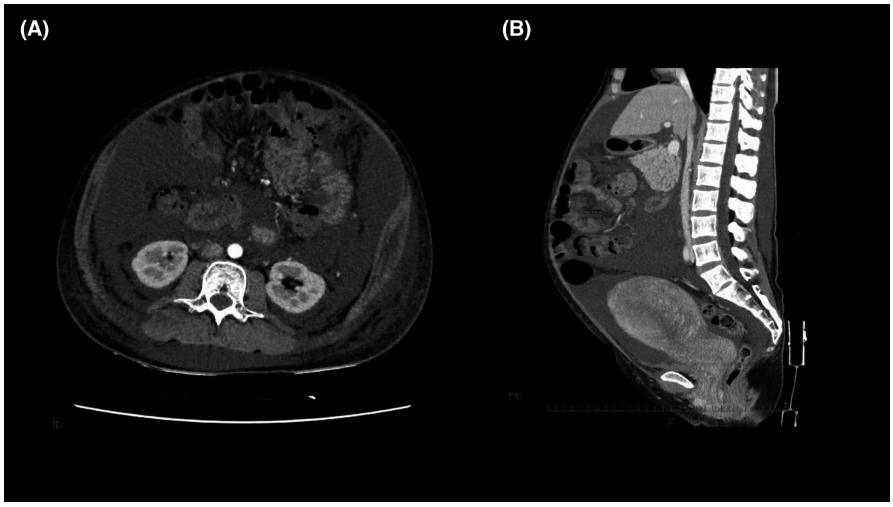


**FIGURE 1** Computed tomography demonstrating the accumulation of massive ascites. (A) Transverse plane. (B) Sagittal plane

**TABLE 1** Laboratory data collected in our hospital on Day 2 after delivery

| *Hematology* | | | *Biochemistry* | | |
|---|---|---|---|---|---|
| WBC | 25,700 | /μl | TP | 4.3 | g/dl |
| Neut. | 79.9 | % | Alb | 1.8 | g/dl |
| RBC | 3.18 | $\times 10^6$/μl | AST | 46 | IU/L |
| Hb | 8.9 | g/dl | ALT | 24 | IU/L |
| Platelet | 22.6 | $\times 10^4$/μl | LDH | 395 | IU/L |
| *Coagulation* | | | CK | 350 | IU/L |
| PT | >100 | % | BUN | 22 | mg/dl |
| APTT | 24.5 | s | Cre | 0.97 | mg/dl |
| Fib | 400 | mg/dl | CRP | 3.35 | mg/dl |
| AT-3 | 80 | % | RF | 5.3 | U/ml |
| FDP | 8 | μg/ml | ANA | <40 | |
| *Urinalysis* | | | PR3-ANCA | <1.0 | U/ml |
| P/C ratio | 1.19 | g/g·Cre | MPO-ANCA | <1.0 | U/ml |
| | | | sFlt-1 | 3862 | pg/ml |

**TABLE 2** Features of preeclampsia

| | |
|---|---|
| Severe hypertension | SBP>160 mmHg |
| | DBP>110 mmHg |
| | Taken on 2 occasions at least 4 h apart while on bed rest (unless antihypertensives have been administered) |
| CNS symptoms | Persistent headache not relieved by analgesics |
| | Visual changes |
| Pulmonary edema | Clinically diagnosed |
| Thrombocytopenia | Platelet count <100,000/ml |
| Renal insufficiency | Serum creatinine >1.1 mg/dl, or |
| | doubling of the serum creatinine when other renal diseases have been excluded |
| Liver dysfunction | Increase in liver enzymes to >twice the upper limits of normal |

addition, preeclampsia with massive ascites and respiratory distress due to pleural effusion during pregnancy is a severe condition requiring urgent pregnancy termination.[8] In the present case, massive ascites was observed postpartum two days after delivery. A certain amount of ascites had accumulated before delivery based on a 7-kg weight gain in the final month before delivery.[9] Indeed, the relationship between ascites and rapid weight gain in association with preeclampsia is described.[10] It is necessary to anticipate the development or aggravation of preeclampsia if excessive weight gain is observed. In our case, vascular permeability might be responsible for the gain of 7 kg in late pregnancy. After transvaginal delivery, she received a transfusion of 3120 ml over 6 h following the loss of 980 g of blood with atonic bleeding under the condition of severe endothelial dysfunction. The transfusion she received was only extracellular fluid and oxytocin. The blood products and tranexamic acid were not used because there were no signs of massive bleeding and anemia. A potential fluid overload after delivery by excessive fluid drip may possibly manifest into fluid retention including ascites. In addition, the level of soluble fms-like tyrosine kinase-1 (sFlt-1) in this case was higher in the postpartum period. There are no diagnostic criteria for sFlt-1 values, but levels of sFlt-1 gradually decrease after delivery and stabilized around 77 pg/ml.[4] In this case, it was suggested that sFlt-1 was high at the time of delivery. sFlt-1 is an important factor in microvascular damage. The model suggests elevated sFlt-1 and associated microvascular damage may be severe during delivery and ascites was likely to accumulate.

The principal clinical finding in the present case is massive ascites predominant to other fluid retention in the postpartum period. Koseoglu et al. reported a case of severe preeclampsia that developed massive ascites and pleural effusion three days after cesarean section, which is somewhat analogous to the present case. In their case, acute postpartum ascites was quite predominant to pleural effusion. They concluded that the cause of massive ascites in the postpartum period is difficult to identify; however,

the obstetrician must pay attention to severe preeclampsia associated with massive ascites in postpartum.[11] Although we also do not know the exact reason for the predominant massive ascites accumulated in postpartum, elevated intra-abdominal pressure (IAP) in antepartum may be a key physiological component for resolving this issue. Chun and Kirkpatrick[12] describe how term pregnancy and immediate postpartum phases may be associated with elevated IAP, which drops significantly after delivery. Intra-abdominal hypertension (IAH) is defined as a sustained IAP greater than 15 $cmH_2O$. Antepartum IAP levels are thought to be significantly higher in patients with pre-eclampsia.[13] The abdominal cavity can be considered as a semi-closed compartment, and any volume change in abdominal content can affect IAP. An excessive elevated IAP before delivery and a significant dropped IAP after delivery might contribute to the predominant massive ascites two days after delivery. However, one may wonder how a majority of preeclampsia does not show such a predominant massive ascites after delivery. In terms of this question, we do not have an exact answer. Yet, it is possible that a predominant massive ascites may develop if a rapid aggravation of preeclampsia and reduction in IAP sequentially overlap. Responding to the rapid reduction of IAP levels after delivery, a large amount of ascites might have leaked into the abdominal cavity two days after delivery.

There are several reports relating management of PE with massive ascites. One cohort study reported that ascites was associated with maternal events such as eclampsia, pulmonary edema, renal failure, and disseminated intravascular coagulation.[5] A second prospective cohort study showed an association between ascites and both maternal and perinatal outcomes such as premature obstetrics and low birthweight.[14] There is no clear etiology of ascites with PE, as well as no clear evidence for the management of PE associated with ascites. According to one report, early termination may improve the outcome in cases of preeclampsia associated with ascites.[15] Based on the existing literature, earlier termination should be considered to improve both maternal and infant outcomes if obvious ascites is observed with preeclampsia.

In this case, isolated proteinuria and a 7-kg weight gain were observed in the third trimester, but blood pressure remained normal. It was reported that 13.7% of pregnant women diagnosed with isolated proteinuria developed PE during pregnancy and 8.4% developed PE postpartum.[16] In addition, pregnant women who developed isolated proteinuria 33 weeks after pregnancy were reported to have high blood pressure at the end of pregnancy.[2] Another report found pregnant women with new-onset isolated proteinuria were more likely to develop PE, and the risk of fetal growth retardation, HELLP syndrome, and neonatal complications will be increased.[1] These observations suggest that isolated gestational proteinuria that progresses to PE is mainly associated with late-onset PE. We suggest close monitoring of these pregnant women will result in favorable maternal and neonatal results.

## 4 | CONCLUSIONS

Postpartum massive ascites associated with preeclampsia is rare, and the mechanism of ascites accumulation is not understood. In this case, proteinuria in the absence of hypertension, the important risk factor for late-onset PE, proceeded with postpartum ascites accumulation. Therefore, when isolated proteinuria is detected in pregnancy, we must consider the potential diagnosis of severe PE including massive ascites accumulation, and its associated adverse outcomes for mother and infant, and manage carefully.

### AUTHOR CONTRIBUTIONS

SH, EK, ES, TU, TS, SA, TK, HM, and SA actively involved in the clinical care of patients. SF, MH, RU, and TM searched the literatures. SH, EK, and ES wrote the manuscript. KY revised the manuscript.

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### CONFLICT OF INTEREST

The authors declare no conflict of interest.

### DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

### CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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