

Received: 2026.06.08
Accepted: 2026.09.09
Available online: 2026.09.22
Published: 2026.XX.XX

Maternal Ascites as an Atypical Presentation of Severe Preeclampsia: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABDEF **Kylie Rayburn** 
ADE **Jesse Cottrell** 
A **Amanda Pauley**

Department of Obstetrics and Gynecology, Marshall University Joan C. Edwards School of Medicine, Huntington, WV, USA

Corresponding Author: Kylie Rayburn, Department of Obstetrics and Gynecology, Marshall Joan C. Edwards School of Medicine, 1600 Medical Center Drive, Huntington, WV 25701, USA
e-mail: johnson1317@marshall.edu
Financial support: None declared
Conflict of interest: None declared

Patient: Female, 23-year-old
Final Diagnosis: Preeclampsia
Symptoms: RUQ pain
Clinical Procedure: —
Specialty: Obstetrics and Gynecology

Objective: Unusual clinical course
Background: Preeclampsia is a multisystem hypertensive disorder of pregnancy associated with significant maternal and fetal morbidity. Although third spacing and fluid accumulation may occur in severe disease, maternal ascites is an uncommon manifestation. Recognition of this atypical presentation is important to facilitate timely diagnosis and appropriate management.

Case Report: We present the case of a 23-year-old woman at 30 weeks and 2 days of gestation who developed severe preeclampsia superimposed on chronic hypertension, complicated by marked maternal third spacing and large-volume ascites while admitted for fetal growth restriction with intermittent absent end-diastolic flow on umbilical artery Doppler. Her clinical course was notable for profound capillary leak syndrome with significant fluid accumulation, including ascites (1.8 L) and pleural effusions. The extent of third spacing represented an unusual manifestation of severe hypertensive disease and created additional diagnostic and management considerations. Careful clinical assessment and recognition of the relationship between her fluid accumulation and severe preeclampsia were essential to guiding management and determining the need for delivery in the setting of maternal and fetal risk.

Conclusions: Severe preeclampsia may present with atypical manifestations beyond the classic features of hypertension and proteinuria. Profound capillary leak with marked third spacing, large-volume ascites, and pleural effusions can occur and may complicate the clinical evaluation of affected patients. Awareness of the uncommon presentation may facilitate earlier recognition of disease severity and prevent delays in management. In patients with severe hypertensive disease and unexplained fluid accumulation, preeclampsia should remain an important consideration. Prompt recognition of maternal or fetal deterioration and timely escalation of care are critical to optimizing outcomes and expediting delivery when clinically indicated.

Keywords: Ascites • Delivery, Obstetric • Fetal Monitoring • Obstetrics • Pre-Eclampsia

Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/954392>

 1659  1  3  6



Publisher's note: All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article, or claim that may be made by its manufacturer, is not guaranteed or endorsed by the publisher

Introduction

Preeclampsia is a multisystem disorder of pregnancy characterized by new-onset hypertension with evidence of end-organ dysfunction after 20 weeks of gestation and remains a leading cause of maternal and perinatal morbidity worldwide [1]. Severe disease may involve neurologic, renal, hepatic, hematologic, pulmonary, and placental complications [1]. Although generalized edema is common in pregnancy, clinically significant abdominal ascites is rare and associated with severe disease and adverse maternal-fetal outcomes [2].

The pathophysiology of preeclampsia involves abnormal placentation, widespread endothelial dysfunction, and angiogenic imbalance leading to increased vascular permeability and intravascular fluid shifts [3]. Elevated levels of soluble fms-like tyrosine kinase-1 (sFlt-1) antagonize vascular endothelial growth factor and placental growth factor (PlGF), contributing to endothelial injury and capillary leak [3,5]. The sFlt-1/PlGF ratio has emerged as an important biomarker associated with disease severity and placental insufficiency [4]. In rare cases, profound endothelial dysfunction may result in significant third spacing, including pulmonary edema, pleural effusions, and large-volume ascites [2]. In this report, “large-volume” ascites refers to diffuse fluid throughout all 4 abdominal quadrants on imaging with approximately 1.8 L of clear peritoneal fluid evacuated at time of cesarean delivery.

Massive ascites in pregnancy presents a broad differential diagnosis including hepatic disease, nephrotic syndrome, infection, malignancy, ovarian pathology, and intra-abdominal surgical disease. In patients with hypertensive disorders of pregnancy, atypical presentations may delay recognition of worsening preeclampsia, particularly when abdominal pain or imaging findings initially suggest hepatobiliary pathology. Gallbladder wall thickening and abdominal fluid accumulation have previously been described in severe preeclampsia as manifestations of systemic capillary leak rather than primary surgical disease [2].

We present a case of severe preeclampsia superimposed on chronic hypertension associated with fetal growth restriction and marked maternal third spacing manifesting as diffuse ascites requiring preterm delivery.

Case Report

A 23-year-old woman, gravida 2 para 0101, was admitted at 30 weeks and 2 days of gestation for inpatient monitoring after umbilical artery Doppler studies demonstrated intermittent absent end-diastolic flow in the setting of fetal growth restriction. Her pregnancy was complicated by chronic hypertension with superimposed preeclampsia, fetal growth

restriction, positive antiphospholipid antibodies (anticardiolipin IgM level of 26), migraines, and pruritic urticarial papules and plaques of pregnancy.

Prior to admission, previous laboratory evaluation demonstrated a urine protein-creatinine ratio of 0.45. She was managed on nifedipine extended release 60 mg twice daily and labetalol 200 mg every 8 hours. Angiogenic marker testing demonstrated a markedly elevated sFlt-1/PlGF ratio of 662, concerning for severe placental dysfunction.

At the time of admission, she received a complete course of antenatal corticosteroids with 2 doses of betamethasone for fetal lung maturation 24 hours apart. Initial inpatient management consisted of close maternal blood pressure monitoring and serial fetal surveillance. On hospital day 3, the patient developed persistent severe-range blood pressures requiring acute antihypertensive therapy with intravenous hydralazine 10 mg. Oral labetalol dosing was subsequently escalated to 600 mg every 8 hours. On the evening of hospital day 4, the patient developed new right upper quadrant abdominal pain. Right upper quadrant ultrasonography demonstrated gallbladder wall thickening, abdominal ascites, and echogenic renal parenchyma. General surgery consultation was obtained due to concern for acute-on-chronic cholecystitis, and broad-spectrum antibiotic therapy with piperacillin-tazobactam was initiated.

On hospital day 5, the patient reported decreased fetal movement. Repeat fetal assessment demonstrated significant deterioration. The biophysical profile score was 2/8, receiving points only for amniotic fluid volume. Umbilical artery Doppler studies showed absent end-diastolic flow with new intermittent reversal (Figure 1). Middle cerebral artery Doppler measured 1.46 multiples of the median, while ductus venosus Doppler remained within normal limits. Ultrasonography additionally demonstrated abdominal ascites in all 4 quadrants and bilateral pleural effusions (Figure 2). A stat chest radiograph demonstrated a small right pleural effusion. Given worsening fetal status in the setting of severe maternal disease, delivery was recommended, and the patient subsequently underwent urgent primary low transverse cesarean delivery. It is important to note, serial laboratory evaluation remained without evidence of HELLP syndrome or significant hepatic or renal dysfunction despite progressive maternal third spacing and worsening fetal status (Table 1).

Upon entry into the abdominal cavity, approximately 1800 mL of clear peritoneal fluid was suctioned, consistent with the large-volume ascites identified throughout all 4 abdominal quadrants on preoperative ultrasonography. Intraoperative evaluation otherwise demonstrated a grossly normal uterus, bilateral fallopian tubes, and ovaries. A viable neonate was delivered, with Apgar scores of 7 and 9 at 1 and 5 minutes,

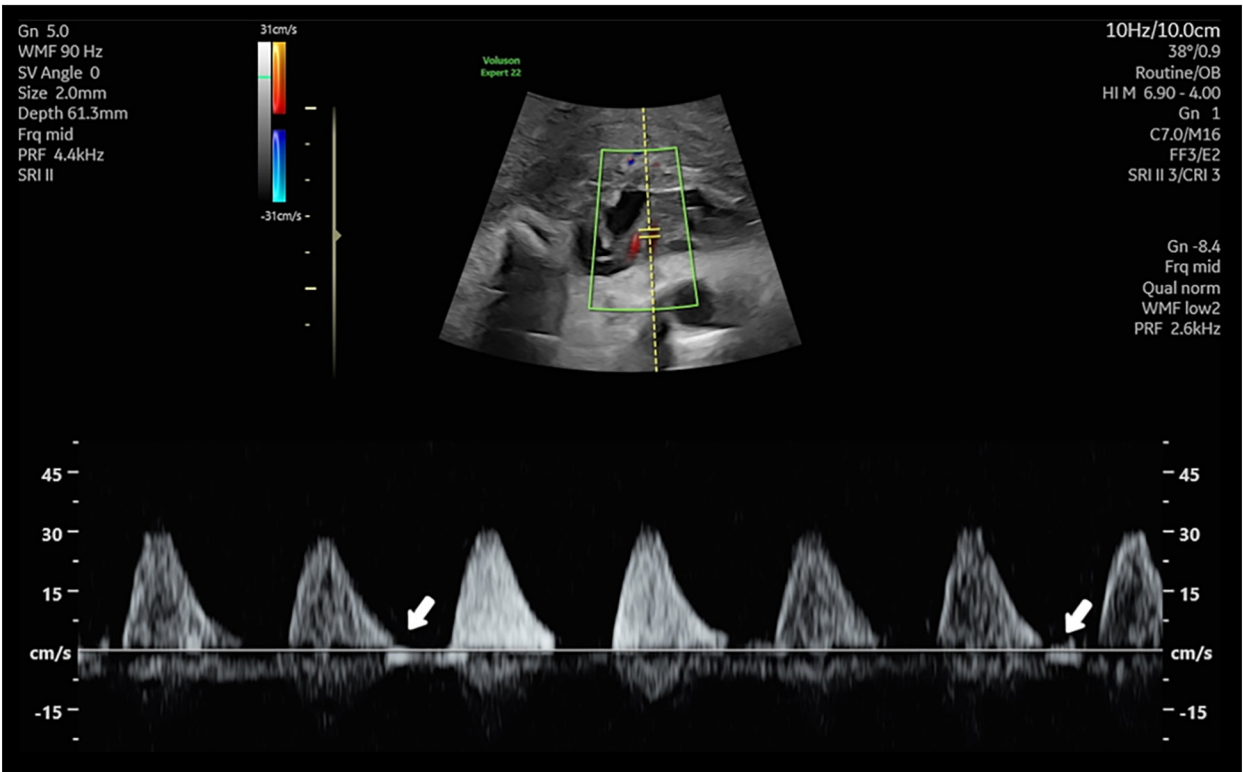


Figure 1. Umbilical artery Doppler velocimetry demonstrating absent and intermittent reversed end-diastolic flow prior to delivery.

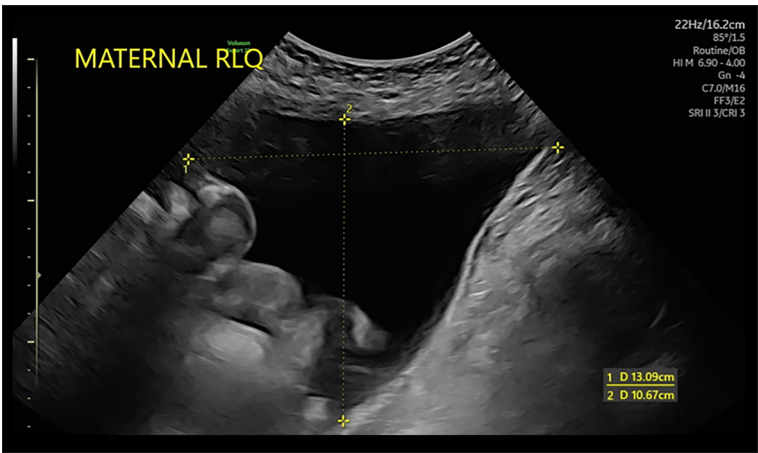


Figure 2. Ultrasound imaging demonstrating maternal abdominal ascites.

respectively, and was admitted to the neonatal intensive care unit for management of prematurity.

Both before and after surgery, the patient received concentrated magnesium sulfate for seizure prophylaxis with serial monitoring. She remained clinically stable on room air without any respiratory compromise. Physical examination demonstrated minimal peripheral edema relative to the degree of abdominal ascites during the cesarean delivery and radiographic third spacing. Computed tomography of the abdomen and pelvis obtained on postoperative day 1 demonstrated moderate-volume abdominal and pelvic ascites, mild generalized anasarca, and

moderate bilateral pleural effusions, with adjacent lower lobe opacities favored to represent atelectatic change rather than pneumonia (**Figure 3**). Following multidisciplinary evaluation, maternal-fetal medicine specialists concluded that the patient's presentation represented severe preeclamptic toxemia with extensive capillary leak and serosal fluid accumulation.

Discussion

This case demonstrates an uncommon and dramatic manifestation of severe preeclampsia characterized by large-volume

Table 1. Longitudinal maternal laboratory evaluation during clinical course.

Laboratory	Admission	HD3	HD4	Delivery	POD1	Ref range
Protein/creatinine ratio	0.45	—	—	—	—	(≤ 0.3)
sFlt-1/PlGF ratio	662	—	—	—	—	(< 38)
Hemoglobin	10.5	10.4	9.5	10.9	7.9	(12.0-16.0)
Platelet count	208	221	210	265	230	(150-440)
AST	22	28	25	24	21	(13-39)
ALT	13	29	26	29	19	(7-52)
LDH	231	248	237	246	246	(140-271)
Creatinine	0.64	0.82	0.73	0.95	0.98	(0.6-1.2)
BUN	15	24	22	22	21	(7-25)
Uric acid	7.4	8.7	8.8	8.8	8.7	(2.3-6.6)
White blood cell count	12.7	16.61	15.36	15.85	26.02	(4.5-10.0)

Abbreviations: sFlt-1, soluble fms-like tyrosine kinase-1; PlGF, placental growth factor; AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; BUN, blood urea nitrogen.



Figure 3. Computed tomography of the abdomen and pelvis on postoperative day 1 demonstrating moderate-volume ascites and bilateral pleural effusions (white arrows).

ascites. Although edema is common in hypertensive disorders of pregnancy, clinically significant maternal serosal effusions are relatively rare and are typically associated with severe endothelial dysfunction and advanced disease [1,2].

The pathogenesis of ascites and pleural effusions in preeclampsia is thought to involve widespread endothelial injury leading to increased vascular permeability, reduced plasma oncotic pressure, and intravascular volume depletion with third spacing [3]. Severe placental dysfunction further contributes through angiogenic imbalance, particularly elevated antiangiogenic factors such as sFlt-1 [3,5]. In this patient, the markedly elevated sFlt-1/PlGF ratio of 662 strongly supported severe

placental disease and correlated with progressive maternal and fetal deterioration. Prior studies have demonstrated that elevated sFlt-1/PlGF ratios are strongly associated with adverse maternal outcomes and severe placental insufficiency [4].

Although generalized third spacing is a recognized manifestation of severe preeclampsia, large volume ascites represents a much less common expression of the same underlying endothelial dysfunction. Third spacing may manifest as peripheral edema, pulmonary edema, pleural effusions, gallbladder wall edema, or smaller volume intraperitoneal fluid collections. In contrast, extensive ascites producing diffuse 4 quadrant abdominal fluid accumulation and approximately 1800 mL of intraoperative drainage, as seen in our patient, has only been reported rarely. Published reports suggest patients with significant ascites may have more severe maternal disease and increased risks of complications such as pulmonary edema, HELLP, and eclampsia [6]. However, these complications did not develop in our patient, who remained clinically stable from a respiratory standpoint, despite marked radiographic and operative evidence of serosal fluid accumulation.

The patient's presentation posed a diagnostic challenge because her right upper quadrant pain and gallbladder wall thickening were nonspecific findings that overlapped with the clinical presentation of her underlying condition. Gallbladder wall edema has previously been reported in severe preeclampsia and may reflect systemic endothelial dysfunction and capillary leak rather than primary biliary disease [2]. Similarly, ascites identified on imaging prompted concern for intra-abdominal

pathology before recognition of worsening preeclamptic toxemia. This highlights the importance of maintaining a broad differential diagnosis while recognizing atypical manifestations of hypertensive disease in pregnancy.

The coexistence of fetal growth restriction with absent and subsequently reversed end-diastolic flow reflected severe placental insufficiency. Progressive fetal compromise was evidenced by decreased fetal movement, a biophysical profile score of 2/8 (points only for amniotic fluid), and worsening umbilical artery Doppler abnormalities. These findings, in conjunction with maternal clinical deterioration and radiographic evidence of widespread third spacing, prompted expedited delivery.

Massive ascites in preeclampsia has been associated with increased maternal morbidity, including pulmonary edema, respiratory compromise, renal dysfunction, and progression to HELLP syndrome or eclampsia [1,2]. Interestingly, despite substantial intra-abdominal and pleural fluid accumulation, this patient remained clinically stable from a respiratory standpoint, with preserved oxygen saturation and reassuring cardiopulmonary examinations. This discrepancy underscores the importance of imaging and fetal surveillance in evaluating disease severity even when overt maternal symptoms are limited.

Delivery remains the definitive treatment for severe preeclampsia [1]. In this case, prompt recognition of worsening fetal status and evolving maternal systemic involvement allowed timely intervention before progression to overt respiratory failure or eclampsia. Postpartum persistence of ascites and pleural effusions further supports the degree of endothelial injury and capillary leak associated with advanced disease.

This case contributes to the limited literature describing severe preeclampsia with massive maternal ascites and emphasizes the need for heightened awareness of atypical presentations. Early recognition can facilitate multidisciplinary management and appropriate timing of delivery to optimize maternal and neonatal outcomes.

References:

1. Sibai BM. Diagnosis and management of gestational hypertension and preeclampsia. *Obstet Gynecol.* 2003;102(1):181-92
2. Vaijyanath AM, Nalliah S. Massive ascites in severe pre-eclampsia: A rare complication. *BMJ Case Rep.* 2012;2012:bcr2012006505
3. Rana S, Lemoine E, Granger JP, Karumanchi SA. Preeclampsia: Pathophysiology, challenges, and perspectives. *Circ Res.* 2019;124(7):1094-112
4. Zeisler H, Llorba E, Chantraine F, et al. Predictive value of the sFlt-1: PlGF ratio in women with suspected preeclampsia. *N Engl J Med.* 2016;374(1):13-22
5. Thadhani R, Mutter WP, Wolf M, et al. First trimester placental growth factor and soluble fms-like tyrosine kinase 1 and risk for preeclampsia. *J Clin Endocrinol Metab.* 2004;89(2):770-75
6. Higami S, Kondo E, Shibata E, et al. A case of preeclampsia developing massive ascites after delivery. *Clin Case Rep.* 2022;10(7):e05830

Conclusions

This case highlights that large-volume ascites and pleural effusions can be uncommon manifestations of severe preeclampsia and can initially mimic alternative intra-abdominal pathology. In similar clinical presentations, progressive serosal fluid accumulation should prompt careful reassessment for worsening hypertensive disease alongside consideration of other potential etiologies. Although this single case cannot be generalized, it emphasizes that diagnostic evaluation and management should be guided by the overall maternal-fetal clinical picture, with timely delivery when maternal and/or fetal status deteriorates.

Acknowledgements

All authors included declare that this manuscript is original, has not been published before, and is currently not being considered for publications elsewhere. We confirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfy criteria for authorship who are not listed. We understand that the Corresponding Author is the sole contact for the Editorial process. She is responsible for communicating with the other authors about progress, submissions of revisions, and final approval of proofs.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report, including accompanying images, and is available upon request.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.