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CASE REPORT PEER REVIEWED | OPEN ACCESS

A series of unfortunate events: Eclampsia with massive post-partum ascites

Spogmai Saeed Khan, Sher Naidoo Roalkvam, Albert De Ridder Harmse, Yamine Saddouk

ABSTRACT

Postpartum ascites in preeclampsia and eclampsia is a rare complication associated with increased maternal morbidity and mortality. Here, we present a case of postpartum ascites, primarily localized in the gastrointestinal interstitium. Medical management with intravenous albumin to increase oncotic pressure, with piggybacked intravenous diuretics to facilitate fluid removal, showed significant clinical improvement.

Keywords: Eclampsia, Interstitial edema, Postpartum ascites, Pre-eclampsia, Pregnancy

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INTRODUCTION

Preeclampsia is defined as new-onset hypertension with clinical signs of organ dysfunction presenting at >20 weeks of gestation [1]. The development of one or more tonic-clonic seizure in a patient with preeclampsia is called eclampsia [2]. Eclampsia is on the severe end of the disease spectrum that is associated with increased fetomaternal morbidity and mortality [2–4]. In our case, we aim to elucidate a rare complication of massive postpartum ascites in the setting of eclampsia presenting as extravasated fluid primarily within the interstitium of the gastric and intestinal walls along with its medical management.

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CASE REPORT

A 32-year-old, gravida 3, para 2, aborta 0, Caucasian female with history of intrauterine growth restriction during second pregnancy and no past medical history presented at 37.1 weeks of gestation presented to ER after a seizure episode. She received prenatal care from a midwife with the last visit at 23.5 weeks gestation was brought to the ER. She had complaints of headache overnight and subsequently had an episode of seizure. In the ER, the patient was postictal but followed one step command. Vitals revealed a blood pressure of 160/116 mmHg, heart rate of 71 bpm, respiratory rate of 22 bpm with an oxygen saturation of 98% on room air. On examination, the patient is a well-nourished young female, somnolent, alert, and oriented x1, her extremities showed deep tendon reflex of 3+. Her uterine fundus measured 35 cm in height. The rest of the physical exam was unremarkable. Lab values are mentioned in Table 1. She was given a 10 mg hydralazine, and 2 mg magnesium initiated. Ultrasound (US) abdomen revealed single live intrauterine pregnancy with ultrasound estimated fetal age of 34 weeks 3 days in vertex presentation with fetal heart rate (HR) of 150 bpm. Placenta was fundal. No previa noted. Amniotic fluid index equals 7.5 cm. No free fluid was noted on this US. An emergent C-section was performed and healthy male fetus was delivered.

On post-operative day (POD) 1, the patient remained altered. On exam, she was somnolent and had a soft abdominal with mild tenderness at the scar site. No guarding or distension noted, with palpable uterine fundus.

Table 1: Serum chemistries including CBC, CMP, magnesium, phosphates, uric acid, coagulation profile, and urine studies

	Admission date	POD-1	POD-2	POD-3	POD-4
Hematology					
WBC (per 10^3 mm^3)	24.0	14.0	17.1	14.6	19.7 H
Hemoglobin (g/dL))	3.7	2.7	2.6	2.2	2.8
Hematocrit (%)	11.2	8.7	7.8	8.6	7.9
Platelets (per µL)	199	149 |	178	154	126
Coagulation					
PT	9.7				11.1
INR	0.9				1.0
APTT	26.9				
Firbrinogen	446				489
D-Dimer					5.43
Chemistry					
Sodium	132	133	130	134	140
Potassium	4.0	4.2	4.5	4.1	3.3
Chloride	104	104	105	109	113
Carbon Dioxide	15.0	21.0	22.0	19.0	21
Creatinine	0.86	0.72	0.97	0.88	0.61
Estimated Creatinine Clearance	81	97	72	79	114
Est GFR	>60	>60	>60	>60	>60
BUN/Creatinine ratio	14	21	27	28	21
Glucose	143	92	89	84	86
Lactic acid	5.4		0.4		
Uric acid	8.1				
Calcium	8.5	7.8	7.2	6.7	6.6
Phosphorus	5.1	4.9		3.5	
Magnesium Repeat Mg	2.8 (+) 8.8	4.2	3.3	3.5	2.7
Total bilirubin	0.5	0.5	0.6	0.8	0.61
AST	46	51	45	35	26
ALT	20	24	26	22	19
Alkaline phosphatase	164	136		110	102
Lactate dehydrogenase	543			265	
Total protein	6.2	5.6	5.6	5.7	5.3
Albumin	2.0	1.8	1.8	1.8	2.4
Globulin	4.2	3.8	3.8	3.3	2.9
Albumin/Globulin ratio	0.5	0.5	0.5	0.2	0.8
Urine				Light-yellow	
Urine color	Yellow			Clear	
Urine clarity	Clear			5.5	
Urine pH	6.5			1.020	
Urine specific gravity	1.032			1+	
Urine protein	3+			Negative	
Urine glucose	1+			1+	
Urine ketones	Trace			2+	

Table 1: *(Continued)*

	Admission date	POD-1	POD-2	POD-3	POD-4
Urine blood	3+			Negative	
Urine nitrates	Negative			Negative	
Urine bilirubin	Negative			Normal	
Urine urobilinogen	Normal			Negative	
Urine leukocyte Estrase	Negative			0–3	
Urine RBC	4–20			5–10	
Urine WBC	0–4			None	
Urine squamous epithelial cells	0-10			Rare	
Urine bacteria	Rare				
Hyaline casts	98			None Seen	
Urine mucus	Rare			81.5	
Urine random creatinine	125			77.2	
Urine random total protein	2025.2			7	
Urine random sodium					
Protein/creatinine ratio	16.2				

ALT = alanine aminotransferase; AST = aspartate aminotransferase; BUN = blood urea nitrogen; LDH = lactate dehydrogenase; POD = postoperative day; PT = prothrombin time; PTT = partial thromboplastin time; WBC = white blood cell.

- CT brain: Abnormal low attenuation in the brainstem, cerebral peduncle, and white matter of the right posterior occipital lobe.
- MRI and MRA brain: findings consistent with extensive posterior reversible encephalopathy syndrome (PRES).

POD-2, the patient complained of dull diffuse abdominal pain. She was noted to have significant abdominal distension and tenderness.

- US pelvis: Involuting uterus with moderate intra-abdominal fluid.
- CT abdomen and pelvis: A large amount of ascites and severe interstitial edema of the gastric and intestinal walls. Small bilateral pleural effusions were also noted (Figure 1).
- Transthoracic echocardiogram: small pericardial effusion.
- Chest X-ray: Bilateral edema/infiltrates and pleural effusions. Left lower lobe consolidation, and cardiomegaly.

Urine output decreased to less than 30 cc/h. Although, computed tomography (CT) abdomen showed worsening abdominal ascites but the ultrasound failed to identify a safe pocket for drainage via paracentesis. The majority of ascitic fluid was confined to the walls of the visceral mucosa. No demonstrable organic cause, other than pre-eclampsia could be found to explain the ascites.

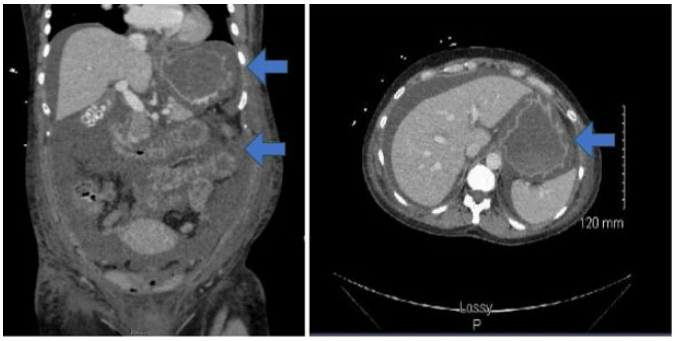

Figure 1: Severe interstitial edema of the gastric and intestinal walls; arrowheads marking the area of interstitial edema.

POD-3, the lab testing continued to show significant persistent proteinuria 3+, urine random total protein of 2000 mg/dL, and protein/creatinine ratio of 16.2. Serum albumin was low at 1.8 g/dL.

We hypothesized that increasing her intravascular oncotic pressure with intravenous (IV) albumin with piggybacked IV diuretics with bumetanide would facilitate fluid removal. Following this intervention, her urine output increased to 250 cc/h and her abdominal distention resolved over the next 48 hours. She was weaned off IV blood pressure medications and transitioned to oral labetalol. She was discharged home on day 7 of her hospitalization. Flowchart in Figue 2 summarizes the patient management while inpatient.

Follow-up at three months showed a complete resolution of all her neurological symptoms and an uneventful postpartum course.

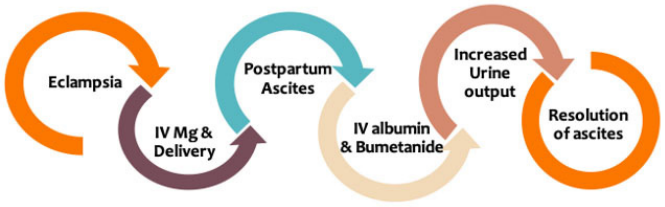

Figure 2: Flowchart denoting the management of patient.

DISCUSSION

Eclampsia is defined as the occurrence of one or more generalized, tonic-clonic convulsions unrelated to other medical conditions in women with hypertensive disorder of pregnancy [2]. The estimated prevalence of pre-eclampsia and eclampsia globally is 4.6% and 0.3%, respectively [5]. The exact pathophysiology of the disease spectrum is unknown; however, endothelial damage and decreased oncotic pressure resulting in generalized capillary, and alteration of autoregulation in the cerebral circulation are the proposed mechanisms [2, 3, 6, 7].

Antenatal ascites in preeclamptic women have been reported in numerous case reports [3]. 8 in 1000 cases during pregnancy develop ascites which develop between 27 and 31weeks of gestation [8]. Preeclamptic/eclamptic patients are at risk of limited literature that describes the incidence of massive ascites in preeclamptic patients during the postpartum period [9]. As per our literature review, only one case of post-partum ascites associated with etiopathology of preeclampsia has so far been reported [10]. The development of post-partum ascites is hypothesized to be due to the sudden change in intra-abdominal pressure (IAP) from the involuting uterus resulting in negative IAP [10]. This, in addition to reduced intravascular oncotic pressure and endothelial damage results in trans-endothelial movement of fluid into the intra-abdominal space resulting in abdominal distention.

Our case is unique in its presentation because the majority of the extravascular volume was located within the mucosa and interstitial walls of the stomach and small intestine. Paracentesis was not a consideration because of the absence of a safe window of approach, medical management by increasing the intravascular oncotic pressure with IV albumin, followed by IV diuresis with bumetanide that facilitated volume removal. Within 24 hours, urine output increased and abdominal distention improved significantly.

There is no evidence-based guideline that describes the management of postpartum ascites associated with preeclampsia or eclampsia [3]. Various case studies have reported a wide range of approach for management from watchful waiting to aggressive intervention via paracentesis and even exploratory laparotomy. [3, 6–12]. Additionally, underlying etiology should be the focus of management to prevent recurrence of this complication. Continued postpartum hypertension should be administered a long-acting oral antihypertensive agents, i.e., labetalol and nifedipine that are compatible with breastfeeding [13]. Women with a history of eclampsia are at increased risk of preeclampsia in a subsequent pregnancy [2]. Additionally, low-dose aspirin (dosage ranging 60–150 mg daily) has been proven to reduce the risk of preeclampsia by 10%to 15% [14].

The paucity of reports in the literature regarding massive postpartum ascites may be due to under reporting. This can be a daunting situation for new mothers and their families. To facilitate healthcare professionals in clinical decision making, reduce patient distress, length of stay, and better clinical outcomes, further data collection and controlled trials are required to prevent recurrence in future pregnancies.

CONCLUSION

Antepartum eclampsia can manifest wide range of postpartum complications. Massive ascites is a rare complication but can present as gastrointestinal interstitial edema. We recommend intravascular albumin infusion followed by diuretic therapy that facilitates raising intravascular oncotic pressure and offloading interstitial fluid, respectively.

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Author Contributions

Spogmai Saeed Khan – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Sher Naidoo Roalkvam – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Albert De Ridder Harmse – Interpretation of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Yamine Saddouk – Interpretation of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.