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Perinatal outcomes in severe preeclampsia with peritoneal ascites – a single tertiary center experience

Perinatální výsledky u těžké preeklampsie s peritoneálním ascitem – zkušenosti z jednoho terciárního centra

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Summary: **Aim:** This study aimed to evaluate maternal and neonatal outcomes of women with severe preeclampsia (PE) with and without peritoneal ascites. **Methods:** In this retrospective cohort study, 76 pregnant women diagnosed with severe PE between January 2019 and January 2024 were evaluated, including 23 with peritoneal ascites and 53 without. Maternal demographic, clinical, and laboratory parameters including neonatal outcomes were compared between the two groups. **Results:** Women with severe PE and peritoneal ascites had higher rates of previous preeclampsia (30.4 vs. 7.5%; P = 0.009), visual symptoms (52.2 vs. 22.6%; P = 0.011), and maternal blood transfusion (47.8 vs. 13.2%; P = 0.001). They also exhibited significantly higher urea levels and proteinuria (P < 0.05). In terms of neonatal outcomes, the ascites group delivered earlier (31.5 ± 3.9 vs. 33.8 ± 3.1 weeks; P = 0.008), had lower birth weights (median 1,400 g vs. 1,990 g; P = 0.015), and higher NICU admission (60.9 vs. 34.0%; P = 0.029) with longer NICU stays (P = 0.003). One case of HELLP syndrome was observed in the ascites group. **Conclusion:** Presence of peritoneal ascites in severe PE is associated with worse maternal and neonatal outcomes, including increased transfusion requirement and adverse perinatal indicators. Detection of ascites may serve as an important clinical marker of disease severity, highlighting the need for closer surveillance and timely intervention.

Key words: blood transfusion – perinatal outcome – peritoneal ascites – pregnancy – severe preeclampsia

Summary: **Cíl:** Tato studie si kladla za cíl zhodnotit mateřské a novorozenecké výsledky u žen s těžkou preeklampsií (PE) s peritoneálním ascitem a bez něj. **Metody:** V této retrospektivní kohortové studii bylo hodnoceno 76 těhotných žen s diagnózou těžké PE v období od ledna 2019 do ledna 2024, z toho 23 s peritoneálním ascitem a 53 bez něj. Mezi oběma skupinami byly porovnány demografické, klinické a laboratorní parametry matky, stejně jako novorozenecké výsledky. **Výsledky:** Ženy s těžkou PE a peritoneálním ascitem měly vyšší míru předchozí preeklampsie (30,4 vs. 7,5 %; p = 0,009), zrakových symptomů (52,2 vs. 22,6 %; p = 0,011) a transfuzí krve matky (47,8 vs. 13,2 %; p = 0,001). Vykazovaly také významně vyšší hladiny močoviny a proteinurii (p < 0,05). Z hlediska neonatálních výsledků se novorozenci ve skupině s ascitem narodili dříve (31,5 ± 3,9 vs. 33,8 ± 3,1 týdne; p = 0,008), měli nižší porodní hmotnost (medián 1 400 vs. 1 990 g; p = 0,015) a vyšší počet hospitalizací na JIP (60,9 vs. 34,0 %; p = 0,029) s delší hospitalizací na JIP (p = 0,003). Ve skupině s ascitem byl pozorován jeden případ HELLP syndromu. **Závěr:** Přítomnost peritoneálního ascitu u těžké preeklampsie je spojena s horšími mateřskými a neonatálními výsledky, vč. zvýšené potřeby transfuze a nepříznivých perinatálních ukazatelů. Detekce ascitu může sloužit jako důležitý klinický marker závažnosti onemocnění a zdůrazňuje potřebu důkladnějšího sledování a včasné intervence.

Klíčová slova: krevní transfuze – perinatální výsledky – peritoneální ascites – těhotenství – těžká preeklampsie

Introduction

Preeclampsia (PE) is a syndrome marked by the onset of hypertension and proteinuria after the 20th week of gestation or by hypertension and end-organ dysfunction with or without proteinuria [1–3]. In the pathophysiology of preeclampsia, impaired placental angiogenesis in early pregnancy compromises blood flow, decreases oxygen delivery, and induces placental tissue injury. As a result, antiangiogenic

substances are released into the mother's bloodstream. Therefore, it changes the functioning of the mother's overall endothelium system, leading to hypertension and various disease symptoms (including issues with blood, neurological, cardiac, pulmonary, kidneys, and hepatic dysfunction). Consequently, there is a possibility of severe maternal complications, including pulmonary edema, cerebral hemorrhage, liver failure, renal failure, and mortality [4,5]. Fetal/neonatal disease burden may arise from placental hypoperfusion and dysfunction and the resulting need for premature birth [6].

In preeclamptic pregnant women, fluid accumulation in the third spaces such as edema and ascites, pleural effusion, and pericardial effusion may be observed due to decreased plasma osmotic pressure and leakage of proteins into the interstitium secondary to endothelial damage [7–9]. Peritoneal ascites accumulation, one of the serious complications of preeclampsia, may be an important factor in determining the severity and prognosis of the disease. However, the effects of ascites are still not fully understood and there are no clear guidelines.

This study aims to examine the impact of peritoneal ascites on perinatal outcomes in individuals diagnosed with severe preeclampsia.

Material and methods

The study included all pregnant women diagnosed with severe preeclampsia and peritoneal ascites who were managed at the University Hospital between January 1, 2019, and January 1, 2024. The control group comprised pregnant women with severe preeclampsia but without peritoneal ascites, matched by mode of delivery. Because in our center almost all women with severe preeclampsia and/or ascites undergo cesarean delivery for maternal – fetal safety, only cesarean deliveries were included to minimize heterogeneity and to allow intraoperative confirmation of ascites. Indications for cesarean delivery included non-reassuring fetal status, severe maternal disease, an unfavorable cervix with urgent need for delivery, and previous uterine scar. Data were collected retrospectively from electronic medical records and archived files. The data was collected retrospectively. The demographic information of all patients was acquired via electronic reports and archive files. The research involving human subjects adhered to all applicable national rules and institutional policies, as well as the principles outlined in the Declaration of Helsinki (updated in 2013). It received approval from the University Ethics Committee under resolution 2023/4667 (16884).

Severe preeclampsia criteria, as defined according to ACOG 2020 criteria, were accepted for the study [1]. Severe preeclampsia was characterized by the presence of at least one of the following indications: Criteria for diagnosis include a systolic blood pressure of 160 mmHg or higher, or a diastolic blood pressure of 110 mmHg or higher on two separate occasions at least 4 hours apart (unless antihypertensive therapy is started before this time). Other indicators include thrombocytopenia (platelet count less than 100×10^9/L), an impaired liver function that cannot be explained by other conditions and is shown by abnormally high levels of liver enzymes in the blood (more than twice the upper limit of normal levels), or severe and persistent pain in the upper right abdomen or upper middle abdomen that does not respond to medication. Additional signs include renal insufficiency (serum creatinine concentration more than 1.1 mg/dL or a doubling of the serum creatinine concentration without other kidney diseases), pulmonary edema, a new-onset headache that does not improve with medication and is not explained by other conditions, and visual disturbances [1]. Ascites was defined as clinically detected and sonographically confirmed free fluid in the peritoneal cavity. The estimated volume was assessed semiquantitatively by ultrasonography; free fluid > 2 cm in the hepatorenal recess, sub-splenic area, or paracolic gutters was classified as pathologic ascites, whereas trace pelvic fluid < 2 cm was considered physiologic. Ascites was additionally confirmed intraoperatively in women undergoing cesarean delivery. The amount of ascites in the peritoneal cavity (mL) was recorded by collecting it with a sterile measuring cup before the Kerr incision was made during the cesarean section. The control group was determined to be cases of severe preeclampsia in which no free fluid was detected during the ultrasonography examination and ascites were not observed during the cesarean section. In our study, cases with multiple pregnancies, pregnancies with fetal anomalies, chronic kidney disease, severe anemia, chronic liver disease, and congestive heart failure were excluded when perinatal outcomes could be significantly worse. Additionally, only cases of cesarean delivery were included. We also excluded patients who did not continue their follow-up in our hospital or gave birth in our clinic from the study. Relevant hematological and biochemical results include systolic blood pressure, diastolic blood pressure, pre- and postoperative hemoglobin levels, white blood cell count, neutrophil, lymphocyte, platelet count, creatinine, uric acid, lactate dehydrogenase, albumin, aspartate aminotransferase, alanine aminotransferase, spot protein/creatinine ratio, and visual symptoms and need for maternal blood transfusion (defined as estimated blood loss of > 1,000 mL after cesarean delivery) were evaluated. Neonatal outcomes examined were gestational age, birth weight, 5-minute APGAR, neonatal intensive care unit (NICU) acceptance rate, and length of stay in the NICU.

We assessed the normal distribution of the data using statistical methods such as Kolmogorov-Smirnov, Shapiro-Wilk tests,

Tab. 1. Comparison of demographic and clinical characteristics in severe preeclampsia with and withou peritoneal ascites.
Tab. 1. Srovnání demografických a klinických charakteristik u těžké preeklampsie s peritoneálním ascitem a bez něj.

Parameters	Severe PE with peritoneal ascites group (N = 23)	Severe PE without peritoneal ascites group (N = 53)	P-value
Age	29.30 ± 7.56	28.13 ± 7.18	0.522[a]
Gravida	2 (1–4)	1 (1–6)	0.961[b]
Parity	0 (0–3)	0 (0–5)	0.497[b]
Abortion	0 (0–3)	0 (0–1)	0.398[b]
BMI	25.29 ± 2.71	25.77 ± 3.61	0.574[a]
IVF pregnancy	3 (13.0%)	5 (9.4%)	0.638[c]
Positive family history of PE	4 (17.4%)	4 (7.5%)	0.099[c]
PE in previous pregnancy	7 (30.4%)	4 (7.5%)	**0.009[c]**
Diabetes mellitus	1 (4.3%)	2 (3.8%)	0.906[c]
Chronic HT	0	1 (1.9%)	0.507[c]
SLE	0	1 (1.9%)	0.507[c]
APAS	0	0	N/A
Thrombophilia	1 (4.3%)	0	0.126[c]
Amount of acid (mL)	700 (100–4,000)		

[a] Independent T-test (Mean ± SD), [b]Mann-Whitney U-test [Median(Min–Max)], [c] Chi-Square test N (%).
APAS – antiphospholipid antibody syndrome, BMI – body mass index, HT – hypertension, IVF – intrauterine insemination, PE – preeclampsia, SLE – systemic lupus erythematosus

and histograms. We utilized independent T-tests or Mann-Whitney U-tests to examine continuous variables, depending on the normality of the distribution. We utilized Chi-square tests or Fisher's exact tests, where applicable, to determine significant differences for categorical variables. All analyses were performed using SPSS 26. Two-tailed $P < 0.05$ was considered statistically significant. For significant comparisons, 95% confidence intervals (CIs) were calculated.

Results

Among the 76 pregnant women diagnosed with severe preeclampsia, 23 (30.3%) had peritoneal ascites, while 53 (69.7%) did not (Tab. 1). Women with severe preeclampsia and ascites were more likely to have had preeclampsia in a previous pregnancy compared to those without ascites (30.4 vs. 7.5%; $P = 0.009$). No significant differences were observed between the groups in terms of age, gravida, parity, abortion history, body mass index (BMI), assisted reproductive technology, family history of preeclampsia, diabetes mellitus, chronic hypertension, systemic lupus erythematosus, antiphospholipid antibody syndrome, or thrombophilia.

Regarding hematological and biochemical parameters (Tab. 2), women with severe preeclampsia and ascites exhibited significantly higher urea levels (median 27.00 mg/dL vs. 22.50 mg/dL; $P = 0.024$) and total urine protein (median 3.00 vs. 1.00; $P = 0.002$) compared to those without ascites. Visual symptoms were more frequent in the ascites group (52.2% [12/23]; 95% CI 32.8–71.2%) than in the non-ascites group (22.6% [12/53]; 95% CI 12.6–36.8%; $P = 0.011$). Maternal blood transfusion was more common in the ascites group (47.8% [11/23]; 95% CI 28.4–67.8%) than in the non-ascites group (13.2% [7/53]; 95% CI 6.4–25.6%; $P = 0.001$).

Birth outcomes (Tab. 3) revealed that women with severe preeclampsia and ascites delivered significantly earlier (the mean gestational age at delivery was lower in the ascites group (31.52 ± 3.94 weeks; 95% CI 29.82–33.22) compared with the non-ascites group (33.81 ± 3.12 weeks; 95% CI 33.03–34.59; $P = 0.008$) and had infants with lower birth weights (median 1,400 g vs. 1,990 g; $P = 0.015$). NICU admission rates were higher in the ascites group (60.9% [14/23]; 95% CI 39.8–79.6%) compared with the non-ascites group (34.0% [18/53]; 95% CI 22.7–47.4%; $P = 0.029$) and these neonates experienced longer NICU stays (median 24 vs. 0 days; $P = 0.003$).

Discussion

In this study, we found that the presence of maternal peritoneal ascites in cases of severe preeclampsia was associated with poorer maternal and neonatal outcomes. Specifically, women with severe preeclampsia with peritoneal ascites had a higher incidence of previous preeclampsia, more frequent visual symptoms, an increased need for maternal blood transfusions, earlier

Tab. 2. Comparative analysis of preoperative and postoperative hematological and biochemical parameters in severe preeclampsia with and without peritoneal ascites.
Tab. 2. Srovnávací analýza předoperačních a pooperačních hematologických a biochemických parametrů u těžké preeklampsie s peritoneálním ascitem a bez něj.

Parameters	Severe PE with peritoneal ascites group (N = 23)	Severe PE without peritoneal ascites group (N = 53)	P-value
Systolic blood pressure	157.09 ± 18.73	156.09 ± 16.75	0.820[a]
Diastolic blood pressure	97.13 ± 13.91	96.51 ± 11.12	0.837[a]
Preoperative Hgb	12.64 ± 1.99	11.89 ± 2.03	0.144[a]
Postoperative Hgb	10.97 ± 1.44	10.90 ± 1.65	0.858[a]
WBC	12.24 ± 3.94	13.17 ± 5.67	0.325[a]
Neutrophil	9.53 ± 3.71	10.55 ± 5.49	0.421[a]
Lymphocyte	2.15 (0.40–4.90)	1.8 (0.10–6.06)	0.270[b]
Platelet	187.48 ± 85.40	223.66 ± 90.91	0.109[a]
Urea	27.00 (11.50–66.80)	22.50 (0.74–89.00)	**0.024[b]**
Creatinine	0.76 (0.33–2.00)	0.60 (0.34–2.10)	0.170[b]
Uric acid	5.77 ± 1.28	6.20 ± 1.39	0.201[a]
LDH	446 (197–1,227)	309 (106–2,250)	0.134[b]
Albumin	30.17 ± 3.98	30.97 ± 4.67	0.478[a]
AST	37.70 (13.70–562.00)	29.00 (9.00–1,248.00)	0.123[b]
ALT	44.00 (8.50–520.00)	19.00 (6.00–972.00)	0.055[b]
TİT Protein	3.00 (0.0–3.00)	1.00 (0.0–3.00)	**0.002[b]**
Spot protein/creatinine	2.10 (0.10–12.00)	2.62 (0.20–6.61)	0.910[b]
Visual symptom	12 (52.2%)	12 (22.6%)	**0.011[c]**
Maternal blood tx	11 (47.8%)	7 (13.2%)	**0.001[c]**
Postpartum HELLP	1 (4.3%)	0	0.126[c]
Eclampsia	1 (4.3%)	0	0.126[c]

[a] Independent T-test (Mean ± SD), [b] Mann-Whitney U-test [Median(Min–Max)], [c] Chi-Square test N (%).
AST – aspartate aminotransferase, ALT – alanine aminotransferase, HELLP – hemolysis, elevated liver enzymes, and low platelet count, Hgb – hemoglobin, LDH – lactate dehydrogenase, PE – preeclampsia

Tab. 3. Neonatal outcomes in severe preeclampsia with and without peritoneal ascites.
Tab. 3. Neonatální výsledky u pacientů s těžkou preeklampsií s peritoneálním ascitem a bez něj.

Parameters	Severe PE with peritoneal ascites group (N = 23)	Severe PE without peritoneal ascites group (N = 53)	P-value
Birth week	31.52 ± 3.94	33.81 ± 3.12	**0.008[a]**
Birth weight	1,400 (520–4,810)	1,990 (590–4,500)	**0.015[b]**
5-minute APGAR score	6 (4–8)	6 (1–8)	0.212[b]
NICU admission (%)	14 (60.9%)	18 (34.0%)	**0.029[c]**
Number of NICU hospitalization days	24 (0–54)	0 (0–48)	**0.003[b]**

[a] Independent T-test (Mean ± SD), [b] Mann-Whitney U-test [Median(Min–Max)], [c] Chi-Square test N (%).
NICU – neonatal intensive care unit, PE – preeclampsia

gestational age at delivery, lower birth weights, and higher rates of NICU admission and longer NICU stays compared to those without peritoneal ascites.

The incidence of preeclampsia is 4.6 percent (95% CI 2.7–8.2) in pregnant women worldwide [10]. Changes in incidence reflect, at least in part, differences in maternal age distribution and the proportion of nulliparous pregnant patients in the population [11]. The prevalence of peritoneal

ascites in preeclampsia reported in the literature varies between 0.8% and 21.6% due to ethnic differences in regions, disease severity, nutritional differences, late presentation for care, and possibly underdiagnosis [12–14]. The plasma colloid osmotic pressure value decreases due to the dilution of blood proteins (mainly albumin and globulins) due to the increase in plasma volume during pregnancy [15]. Abnormalities in placental vascular development early in pregnancy in preeclampsia may result in relative placental underperfusion/hypoxia/ischemia, which may subsequently cause maternal systemic endothelial damage [9]. Since edema and fluid accumulation in the third spaces such as peritoneal ascites, pleural effusion, and pericardial effusion in preeclamptic pregnant women are explained by decreased plasma osmotic pressure and endothelial damage, it is thought that ascites may be an early event in severe preeclampsia [7–9].

One of the most important risk factors for developing preeclampsia is having experienced preeclampsia in the past. Compared to patients without this history, the risk of developing preeclampsia in a subsequent pregnancy increases eightfold (RR 8.4; 95% CI 7.1–9.9) [16]. Studies have shown that patients with preeclampsia who did not have serious features in their first pregnancy had preeclampsia in 5 to 7% of second pregnancies [17,18]. In patients with severe features of preeclampsia in the 2nd trimester, the risk of developing preeclampsia has been reported to be between 25 and 65% [19–22]. In our study, we found that the group with peritoneal ascites in cases of severe PE had a higher history of preeclampsia in their previous pregnancy (30.4 vs. 7.5%; P = 0.009).

In our study, we observed a higher incidence of visual symptoms and the need for a blood transfusion among mothers in the severe PE with peritoneal ascites group compared to those without this condition. Visual symptoms, particularly those related to vision, may occur more frequently in patients with severe PE in the peritoneal ascites group, potentially due to swelling of the optic nerves or retinal detachment [23,24]. In a study, it was observed that there was a greater need for blood transfusion in the severe PE with peritoneal ascites group [25]. In a study including 121 patients in the severe PE with peritoneal ascites, peritoneal ascites was independently associated with adverse maternal outcomes, such as more maternal blood transfusions [26]. In the study where the results of 23 in the severe PE with peritoneal ascites were evaluated, it was decided that the pregnancies could not be followed up because PE in the mother complicated with peritoneal ascites caused serious maternal complications and was terminated [12]. Studies are reporting life-threatening complications in preeclamptic women with peritoneal ascites [13,27]. PE patients with peritoneal ascites are often associated with more severe clinical signs, which may suggest that they may be at greater risk of bleeding and therefore require more blood transfusions. In a large cohort study, it was observed that maternal ascites did not worsen neonatal outcomes in a group of patients with severe preeclampsia [26]. However, in a study including 46 preeclamptic patients diagnosed with antepartum peritoneal ascites, a relationship was found with increased poor neonatal outcomes [28]. In our study, there was no significant difference in the 5-minute APGAR scores between the severe PE with or without peritoneal ascites. In the severe PE group with peritoneal ascites, a significant difference was observed in poor neonatal outcomes, such as an earlier week of birth, a lower birth weight, more NICU acceptance, and NICU length of stay. These results suggest that in severe PE cases, the presence of peritoneal ascites may cause earlier delivery. Although the 5-minute Apgar score is affected by parameters such as premature birth week and low birth weight, medical care and support in our intensive care unit may have eliminated the difference between the 5-minute Apgar scores because it was provided by the same neonatal team.

Severe preeclampsia is associated with a considerable burden of maternal morbidity and mortality. Contemporary evidence indicates that the presence of peritoneal ascites is strongly linked to severe maternal complications, including congestive heart failure, acute respiratory distress syndrome (ARDS), eclampsia, renal failure, disseminated intravascular coagulation (DIC), and even maternal death [29]. In one cohort study, 42% of women with ascites experienced such complications, including four maternal deaths, compared with only 9% among controls [26]. Similarly, in cases of HELLP syndrome, the presence of ascites increased the risk of congestive heart failure sixfold and ARDS ninefold [30]. Unlike studies reporting high rates of severe maternal complications (e. g., ARDS, renal failure, DIC, maternal death), our cohort did not demonstrate these outcomes. This likely reflects our modest sample size, the retrospective design, and our hospital's proactive management protocols. Nonetheless, we observed increased transfusion needs and adverse neonatal outcomes, suggesting that peritoneal ascites remains a marker of disease severity even in the absence of overt catastrophic complications.

Our study has several limitations. First, the retrospective design and single-center setting may limit the generalizability of the findings. Second, our sample size was modest and may be prone to type I or type II errors. Third, because only cesarean deliveries were included, selection bias is possible; however, this approach was deliberately chosen to allow intraoperative confirmation of ascites and to minimize confounding related to the mode of delivery. Additionally, our hospital is a specialized referral center, which may introduce

referral bias. Despite these limitations, our findings may inform clinical practice and guide future research on the management of severe preeclampsia, particularly regarding the role of peritoneal ascites. Prospective, multicenter studies are warranted to further elucidate the impact of ascites on the clinical course of preeclampsia.

Conclusion

This study demonstrates that peritoneal ascites in women with severe preeclampsia is a marker of increased disease severity and is associated with adverse maternal and neonatal outcomes, including higher transfusion requirements, earlier delivery, lower birth weight, and longer NICU stays. Recognition of ascites in preeclamptic patients should alert clinicians to a potentially complicated course, warranting intensified monitoring and proactive management. Further prospective, multicenter studies are required to validate these findings and to clarify whether early identification of ascites could improve maternal and neonatal outcomes.

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