

OBSTETRICS

Cerebral infarcts, edema, hypoperfusion, and vasospasm in preeclampsia and eclampsia



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BACKGROUND: Eclampsia is a serious pregnancy complication and is associated with cerebral edema and infarctions. However, the underlying pathophysiology of eclampsia remains poorly explored.

OBJECTIVE: This study aimed to assess the pathophysiology of eclampsia using specialized magnetic resonance imaging to measure diffusion, perfusion, and vasospasm.

STUDY DESIGN: This was a cross-sectional study recruiting consecutive pregnant women between April 2018 and November 2021 at Tygerberg Hospital, Cape Town, South Africa. Women with eclampsia, preeclampsia, and normotensive pregnancies who underwent magnetic resonance imaging after birth were recruited. The main outcome measures were cerebral infarcts, edema, and perfusion using intravoxel incoherent motion imaging and vasospasm using magnetic resonance imaging angiography. The imaging protocol was established before inclusion.

RESULTS: Here, 49 women with eclampsia, 20 women with preeclampsia, and 10 normotensive women were included. Cerebral infarcts were identified in 34% of women with eclampsia and 5% of women with preeclampsia (risk difference, 0.29; 95% confidence interval, 0.06–0.52; $P=.012$). However, no cerebral infarct was identified in normotensive controls. Women with eclampsia were more likely to have vasogenic cerebral edema than women with preeclampsia (80% vs 20%, respectively; risk difference, 0.60; 95% confidence interval, 0.34–0.85; $P<.001$) and normotensive women (risk difference, 0.80; 95% confidence interval, 0.47–1.00; $P<.001$). Diffusion was increased in women with eclampsia

in the parieto-occipital white matter (mean difference, $0.02 \times 10^{-3} \text{ mm}^2/\text{s}$; 95% confidence interval, 0.00–0.05; $P=.045$) and caudate nucleus (mean difference, $0.02 \times 10^{-3} \text{ mm}^2/\text{s}$; 95% confidence interval, 0.00–0.04; $P=.033$) compared with women with preeclampsia. In addition, diffusion was increased in women with eclampsia in the frontal white matter (mean difference, $0.07 \times 10^{-3} \text{ mm}^2/\text{s}$; 95% confidence interval, 0.02–0.12; $P=.012$), parieto-occipital white matter (mean difference, $0.05 \times 10^{-3} \text{ mm}^2/\text{s}$; 95% confidence interval, 0.02–0.07; $P=.03$), and caudate nucleus (mean difference, $0.04 \times 10^{-3} \text{ mm}^2/\text{s}$; 95% confidence interval, 0.00–0.07; $P=.028$) compared with normotensive women. Perfusion was decreased in edematous regions. Hypoperfusion was present in the caudate nucleus in eclampsia (mean difference, $-0.17 \times 10^{-3} \text{ mm}^2/\text{s}$; 95% confidence interval, -0.27 to -0.06 ; $P=.003$) compared with preeclampsia. There was no sign of hyperperfusion. Vasospasm was present in 18% of women with eclampsia and 6% of women with preeclampsia. However, no vasospasm was present in the controls.

CONCLUSION: Eclampsia was associated with cerebral infarcts, vasogenic cerebral edema, vasospasm, and decreased perfusion, which are not usually evident on standard clinical imaging. This finding may explain why some patients have cerebral symptoms and signs despite having normal conventional imaging.

Key words: eclampsia, hyperperfusion, hypoperfusion, pathophysiology, preeclampsia, vasospasm

Introduction

Preeclampsia is a life-threatening complication of pregnancy that occurs when a woman develops hypertension with end-organ injury in the second half of pregnancy.¹ One of the most common complications of preeclampsia is

eclampsia. Eclampsia presents as generalized tonic-clonic seizures and is associated with cerebral edema in 71% to 80% of cases.^{2–4} The etiology of eclampsia is largely unknown, and there is no direct treatment.

Cerebral edema can be visualized using computerized tomography and conventional magnetic resonance imaging (MRI). It is defined as an increase in brain water content. The common etiologies are cytotoxic (direct cell injury and swelling), vasogenic (blood-brain barrier injury), osmotic (changes in osmolality in plasma or interstitium), and interstitial (increased ventricular pressure). Diffusion-weighted imaging (DWI) is an MRI technique based on differences in random Brownian motion

of water molecules among different tissues. This allows the separation of vasogenic edema and cytotoxic edema.


Advanced MRI techniques can be used to assess cerebral diffusion, perfusion, and vessel vasospasm and to further understand the etiology of edema. Intravoxel incoherent imaging (IVIM) is an advanced DWI technique that enables the extraction of both water diffusion and capillary perfusion.⁵ If cerebral water diffusion is increased, it indicates increased brain water content. Cerebral perfusion can be measured using the IVIM technique by visualizing blood microcirculation in the capillary networks of cerebral tissue.^{5–7} A decreased perfusion indicates insufficient blood flow to a region. To measure

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AJOG at a Glance

Why was this study conducted?

Eclampsia has previously been associated with infarctions on conventional magnetic resonance imaging (MRI). However, this finding needs to be verified in larger prospective studies.

Advanced MRI techniques, including cerebral diffusion (shows subclinical edema), perfusion (capillary blood flow), and angiography, can increase the understanding of the underlying pathophysiology of eclampsia.

Key findings

Eclampsia was associated with cerebral infarcts, increased diffusion, hypoperfusion, and vasospasm.

What does this add to what is known?

Our data provide evidence against hyperperfusion of cerebral arteries, which was previously thought to cause cerebral edema in eclampsia. Of note, women with eclampsia seem to suffer from insufficient cerebral perfusion. This is important knowledge to guide future interventions for neuroprotection. In addition, our study provides robust evidence of the presence of cerebral infarcts that cause potentially irreversible injury after eclampsia.

vasospasm, 3-dimensional (3D) time-of-flight (ToF) magnetic resonance (MR) angiography is used.

Cerebral diffusion, perfusion, and vasospasm have not been studied in preeclampsia and eclampsia. If affected, it may explain the occurrence of neurologic signs and symptoms in women with preeclampsia and eclampsia who do not have evidence of pathology on conventional MRI. In addition, it could offer important insights into the pathophysiology of eclampsia, directing future research.⁸ Therefore, we used these advanced MRI techniques to study women with eclampsia and preeclampsia and normotensive controls.

Materials and methods**Study cohort**

This cross-sectional prospective study included women recruited to the Preeclampsia Obstetric Adverse Events (PROVE) biobank at Tygerberg Hospital, Cape Town, South Africa.⁹ Tygerberg Hospital is the largest referral hospital in the Western Cape Province, with more than 8500 high-risk deliveries a year.⁹

Women were prospectively included after a diagnosis of eclampsia or preeclampsia or at admission for delivery for controls. MRI was performed after

delivery before discharge. We included women who did not have a clinical indication for imaging, such as a suspicion of stroke. Women with known neurologic or cardiac disease were ineligible. For normotensive women, additional exclusion criteria included chronic hypertension and diabetes mellitus. Baseline data were obtained from interview and extraction from medical records. Women were followed up until discharge. Data were recorded on a Research Electronic Data Capture database¹⁰ and double-checked for accuracy.

Exposures

Preeclampsia was defined according to the American College of Obstetricians and Gynecologists (ACOG) Practice Bulletin, but significant proteinuria was also required (protein-to-creatinine ratio of ≥ 30 mg/mmol (0.3 mg/mg) or ≥ 0.3 g of protein in a 24-hour urine collection or urine dipstick of $>1+$ on more than one occasion).¹¹ We only included women with preeclampsia with severe features according to the same classification. This approach was used to distinguish eclampsia as an organ-specific complication from preeclampsia with other severe features.

Eclampsia was defined as generalized tonic-clonic seizures in a woman with preeclampsia in the absence of another etiology. We subdivided eclampsia into eclampsia only (women who had 1 eclamptic seizure with no other neurologic symptom) or complex eclampsia (women who had multiple seizures, a Glasgow Coma Scale of <13 , or eclampsia together with other organ complications).

Pulmonary edema was diagnosed when there was worsening dyspnea, fine bibasal inspiratory crackles on auscultation, and features of pulmonary edema on chest x-ray. Hemolysis, elevated liver enzymes, and low platelet count (HELLP) syndrome was defined as a platelet count of $<100 \times 10^9/L$, aspartate aminotransferase of >70 U/L, and hemolysis (lactate dehydrogenase of >600 U/L or hemolysis on a peripheral blood smear). Renal impairment was defined as a serum creatinine level of >120 $\mu\text{mol/L}$, which is higher than the ACOG definition. Severe hypertension was defined as a systolic blood pressure of ≥ 160 mm Hg and/or a diastolic blood pressure of ≥ 110 mm Hg.

Outcomes measures

MRI was performed at 1.5T (Aera; Siemens Healthineers, Erlangen, Germany). The protocol included sagittal 2-dimensional (2D) T1-weighted spin echo, axial 2D T2-weighted spin echo, axial 2D T2-weighted gradient echo, axial 2D fluid attenuation inversion recovery (FLAIR), and axial diffusion-weighted sequences. The DWI was acquired with 9 b values for the separation of diffusion and perfusion parameters using IVIM.⁵ All assessors were blinded to clinical exposures.

Of the outcomes in our study, only cerebral edema (vasogenic and cytotoxic) can be visualized using conventional MRI protocol.

Acute cerebral infarcts were defined as high-intensity lesions on the b_{1000} DWI with corresponding low-intensity lesions on the apparent diffusion coefficient (ADC) maps. Vasogenic cerebral edema was defined as high-intensity white matter lesions on FLAIR and ADC images and low signal

intensity on DWI. Edema volume was calculated by adding the areas of edema in individual slices, multiplied with slice thickness.

The IVIM parametric maps (D, D*, and f) were calculated from multiple b-value DWI sequence using commercially available software (Olea Sphere 3.0; Olea Medical, La Ciotat, France). These metrics correspond to the rate of water self-diffusion (D), which relates to tissue water content, pseudo-diffusion related to capillary blood flow velocity (D*), and volume fraction of perfused capillaries (f).⁵ D, D*, and f were measured in the frontal white matter, the parieto-occipital white matter, the caudate nucleus, the lentiform nucleus, and the thalamus. Estimates were obtained from averages of the right and left sides in 2 or 3 slices. The same measurements were obtained from regions of edema when present.

Cerebral diffusion was defined as the diffusion coefficient and was used as a sign of subclinical edema. Cerebral perfusion was assessed using pseudo-diffusion and perfusion fraction, yielding the combined perfusion estimate, which correlates with the tissue capillary blood flow.⁵

Vasospasm was defined as a vessel segment with more than 50% diameter reduction compared with adjacent normal-appearing vessels and was evaluated on the 3D ToF MR angiography. The first and second segments of the anterior and posterior cerebral arteries and the first segment of the middle cerebral arteries were analyzed. As this assessment is subjective, independent evaluations were performed by a radiologist and a neuroradiologist who were blinded to clinical data. Segments in which both readers detected more than 50% diameter reduction were assigned positive for vasospasm, not including hypoplastic segments of the first segments of the anterior and posterior cerebral arteries. Further description of MRI sequence parameters can be found in [Supplemental Table 1](#).

Statistical methods

Descriptive data are presented as means with standard deviations or

medians with interquartile ranges (IQRs) for numeric variables and numbers with percentages for categorical variables. Binary variables were analyzed using the Farrington-Manning test to determine the risk difference (RD) between groups. Numeric variables were analyzed using the Welch *t* test, accounting for unequal variances between groups. The results are presented as mean difference (MD) with 95% confidence intervals (CIs). Differences in diffusion and perfusion in edema regions vs parieto-occipital white matter were evaluated using the paired *t* test. Evaluations in subgroups with few observations ($n < 6$) were performed using nonparametric permutation tests, and the corresponding CIs were calculated using test inversion. Positively skewed variables were log-transformed before analysis, and the fold change between groups was calculated by exponentiating the MD on a log scale. All statistical tests were performed at 5% significance level. Statistical analyses were performed using SAS/STAT software (version 9.4 of the SAS system for Windows; SAS Institute Inc, Cary, NC).

Sample size

Previous studies found a large variation in the presence of cerebral infarcts on MRI.^{2,3,12} We aimed for a sample size of 50 women with eclampsia, 20 women with preeclampsia, and 10 normotensive controls to be able to detect a difference in cerebral ischemia between groups. This is similar to other larger prospective studies.^{2,3}

Ethics approval and registration details

Ethics approval was obtained from the Stellenbosch University Health Research Ethics Committee (protocol number: N18/03/034; Federal Wide Assurance number: 00001372; institutional review board number: IRB0005239). All participants or their guardians signed an informed consent. The biobank is registered (registration number: ISRCTN10623443), and the protocol has been published.⁹

Data availability

Anonymized data are available on request after approval by the corresponding author.

Results

Participants

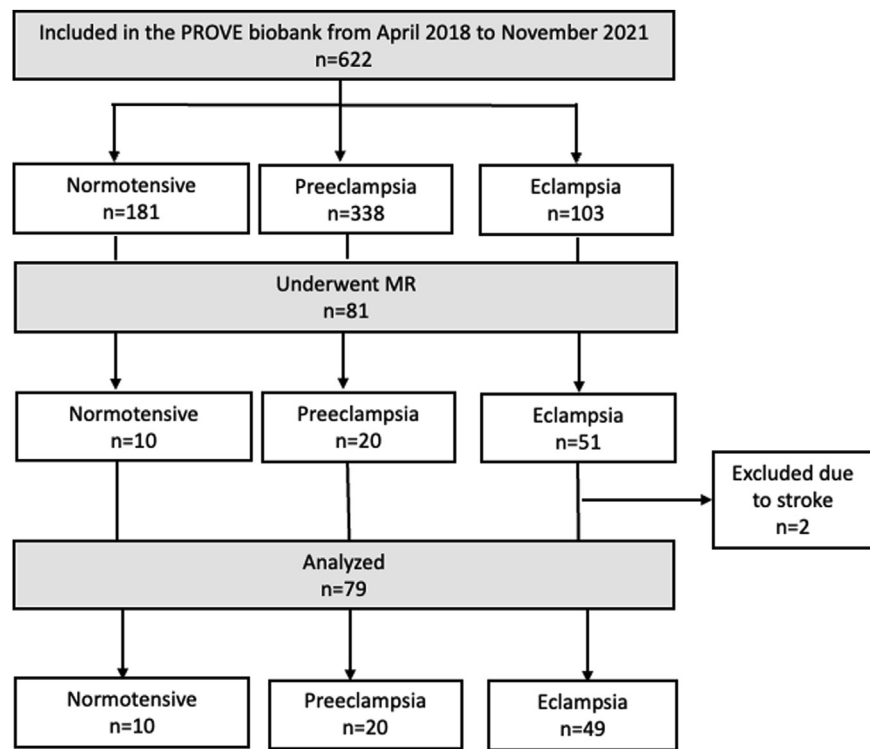
A total of 81 women had MRI examinations between April 2018 and November 2021. The median times from delivery to MRI were 3 days (IQR, 1–6) for women with preeclampsia and 1 day (IQR, 0–13) for women with eclampsia. Of note, 12 women (60%) with preeclampsia and 10 women (20%) with eclampsia had MRI performed more than 48 hours after delivery. In addition, 2 women had clinically evident strokes and were excluded. Furthermore, 41 women had eclampsia, 20 women had preeclampsia without eclampsia, and 10 women had a normotensive pregnancy ([Figure 1](#)).

During the study period, 101 women with eclampsia were included in the PROVE biobank. The most common reason for not performing an MRI was the unavailability of the MRI. There was no major difference in the background characteristics between women who underwent an MRI and those who did not ([Supplemental Table 2](#)).

Maternal characteristics and pregnancy outcomes

Background data can be found in [Table 1](#). Women with preeclampsia and eclampsia were more often nulliparous, less likely to have attended antenatal care, and more likely to experience a stillbirth than normotensive women. In addition, women who experienced eclampsia were more likely to use alcohol and smoke cigarettes during pregnancy ([Table 1](#)). All women with preeclampsia and eclampsia were treated with an antihypertensive medication. All women with preeclampsia had one or more severe features. Of note, 17 women had severe hypertension, 11 women had pulmonary edema, 6 women experienced HELLP syndrome, and 6 women had renal impairment. Moreover, 1 woman with preeclampsia only had headache as a severe feature. Chronic hypertension

FIGURE 1
Flowchart of the study cohort



MR, magnetic resonance; PROVE, Preeclampsia Obstetric Adverse Events.

Bergman. Pathophysiology of eclampsia. *Am J Obstet Gynecol* 2025.

occurred in 5 women (25%) with preeclampsia and 2 women (4%) with eclampsia (Table 1).

Cerebral infarcts

Of note, 16 women (34%) in the eclampsia group had cerebral infarcts compared with 1 woman (5%) in the preeclampsia group (RD, 0.29; 95% CI, 0.06–0.52; $P=.012$) and none (0%) in the normotensive group (RD, 0.34; 95% CI, 0.03–0.65; $P=.030$) (Table 2 and Figure 2).

Vasogenic cerebral edema

Of note, 39 women (80%) in the eclampsia group had vasogenic cerebral edema on MRI compared with 4 women (20%) in the preeclampsia (RD, 0.60; 95% CI, 0.34–0.85; $P<.001$) and none (0%) in the normotensive group (RD, 0.80; 95% CI, 0.47–1.00; $P<.001$) (Table 2 and Figure 2). When vasogenic cerebral edema was present, women with

eclampsia had a larger volume of edema than those with preeclampsia (fold change, 12.20; 95% CI, 3.51–42.70; $P<.001$), as presented in Figure 3.

Systemic blood pressure was not associated with cerebral edema ($P=.99$ for severe hypertension and $P=.51$ for chronic hypertension).

Diffusion (subclinical cerebral edema)

Women with eclampsia had increased diffusion in the frontal white matter (MD, $0.07 \times 10^{-3} \text{ mm}^2/\text{s}$; 95% CI, 0.02–0.12; $P=.012$), the parieto-occipital white matter (MD, $0.05 \times 10^{-3} \text{ mm}^2/\text{s}$; 95% CI, 0.02–0.07; $P=.003$), and the caudate nucleus (MD, $0.04 \times 10^{-3} \text{ mm}^2/\text{s}$; 95% CI, 0.00–0.07; $P=.028$) compared with those with normotensive pregnancies (Table 3). Women with preeclampsia had increased diffusion estimates in the frontal white matter (MD, $0.07 \times 10^{-3} \text{ mm}^2/\text{s}$; 95%

CI, 0.00–0.14; $P=.048$) and a tendency for increased diffusion in both the parieto-occipital white matter (MD, $0.02 \times 10^{-3} \text{ mm}^2/\text{s}$; 95% CI, –0.01 to 0.05; $P=.12$) and caudate nucleus regions (MD, $0.02 \times 10^{-3} \text{ mm}^2/\text{s}$; 95% CI, –0.01 to 0.05; $P=.25$) compared with those with normotensive pregnancies (Table 3 and Figure 4).

Women with eclampsia had increased diffusion in the parieto-occipital white matter (MD, $0.02 \times 10^{-3} \text{ mm}^2/\text{s}$; 95% CI, 0.00–0.05; $P=.045$) and caudate nucleus regions (MD, $0.02 \times 10^{-3} \text{ mm}^2/\text{s}$; 95% CI, 0.00–0.04; $P=.033$) compared with those with preeclampsia (Table 3).

Perfusion

Women with eclampsia had decreased perfusion in the caudate nucleus compared with those with preeclampsia (MD, $-0.17 \times 10^{-3} \text{ mm}^2/\text{s}$; 95% CI, –0.27 to –0.06; $P=.003$) (Table 3 and Figure 4). There was no significant difference between women with eclampsia and normotensive controls.

Vasospasm

Vasospasm was present in 8 women (18.0%) in the eclampsia group compared with 1 woman (5.6%) in the preeclampsia group (RD, 0.13; 95% CI, –0.07 to 0.32; $P=.20$) and none (0%) in the normotensive group (RD, 0.18; 95% CI, –0.06 to 0.43; $P=.14$) (Table 2 and Figure 4).

Diffusion and perfusion in women with cerebral edema

In women with cerebral edema visible on conventional MRI, we compared women with eclampsia with those with preeclampsia. There was a tendency for lower perfusion in women with eclampsia group than in those with preeclampsia (fold change, 0.57; 95% CI, 0.26–1.33; $P=.19$) (Table 4). When comparing edema regions with normal-appearing parieto-occipital white matter, there was an increase in water diffusion (MD, $0.68 \text{ mm}^2/\text{s} \times 10^{-3}$; 95% CI, 0.59–0.76; $P<.001$) and a reduction in perfusion (MD, $-1.00 \text{ mm}^2/\text{s} \times 10^{-3}$; 95% CI, –1.09 to –0.91; $P<.001$) in edema regions (Table 5).

TABLE 1
Baseline characteristics of the study cohort

Characteristics	Normotensive (n=10)	Preeclampsia (n=20) ^a	Eclampsia (n=49)
Demographics and baseline characteristics			
Age (y), mean (SD)	29.5 (6.1)	28.7 (7.7)	22.4 (5.6)
BMI (kg/m ²), ^b mean (SD)	30.0 (6.9)	28.2 (7.6)	24.8 (5.6)
Nulliparous	2 (20%)	8 (40%)	36 (74%)
Any antenatal care	9 (90%)	18 (90%)	41 (84%)
HIV positive	1 (10%)	3 (15%)	6 (12%)
Smoking during pregnancy	2 (20%)	2 (10%)	9 (18%)
Alcohol use during pregnancy	0 (0%)	0 (0%)	7 (14%)
Diabetes mellitus	0 (0%)	0 (0%)	0 (0%)
Chronic hypertension	0 (0%)	5 (26%)	2 (4%)
Mode of birth			
Vaginal	1 (10%)	6 (30%)	17 (35%)
Elective or nonurgent CD	6 (60%)	2 (10%)	1 (2%)
Emergency CD	3 (30%)	12 (60%)	31 (63%)
Gestation at delivery (wk), median (IQR)	38 6/7 (27 3/7 to 41 3/7)	32 6/7 (21 2/7 to 36 1/7)	34 4/7 (24 2/7 to 40 5/7)
Antihypertensive medication at the time of birth	0 (0%)	20 (100%)	49 (100%)
Live-born infant	9 (90%)	14 (70%)	41 (84%)
Birthweight (g), mean (SD)	2729 (842)	1559 (584)	2076 (946)
Maternal complications			
Maternal death	0 (0%)	0 (0%)	0 (0%)
Intensive care unit admission	0 (0%)	1 (5%)	4 (8%)
Eclampsia	0 (0%)	0 (0%)	49 (100%)
Recurrent eclampsia	0 (0%)	0 (0%)	17 (35%)
Intracranial hemorrhage	0 (0%)	0 (0%)	1 (2%)
Glasgow Coma Scale of <13	0 (0%)	0 (0%)	12 (24%)
Cortical blindness	0 (0%)	2 (10%)	2 (4%)
Pulmonary edema	0 (0%)	11 (55%)	1 (2%)
Inotropic support	0 (0%)	1 (5%)	1 (2%)
Renal impairment	0 (0%)	6 (30%)	10 (20%)
Dialysis	0 (0%)	1 (5%)	1 (2%)
HELLP syndrome	0 (0%)	6 (30%)	14 (29%)
Disseminated intravascular coagulation (INR of >1.2)	0 (0%)	1 (5%)	5 (10%)
Severe hypertension	0 (0%)	17 (85%)	17 (35%)
Sepsis	0 (0%)	3 (15%)	4 (8%)
Venous thromboembolism	0 (0%)	0 (0%)	1 (2%)
Placental abruption	1 (10%)	2 (10%)	3 (6%)

Data are presented as mean (SD) or median (IQR) for numeric variables and number (percentage) for categorical variables.

BMI, body mass index; CD, cesarean delivery; HELLP, hemolysis, elevated liver enzymes, and low platelet count; INR, international normalized ratio; IQR, interquartile range; SD, standard deviation.

^a All women had preeclampsia with severe features; ^b Data on BMI is missing for 2 women (20%) in the control group, 8 women (40%) in the preeclampsia group, and 24 women (49%) in the eclampsia group.

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TABLE 2
Vasogenic cerebral edema, infarcts, and vasospasm in normotensive women (controls) and in women with preeclampsia or eclampsia

Variable	RD (95% CI)			
	Normotensive (n=10)	Preeclampsia (n=20)	Eclampsia (n=49)	Preeclampsia vs normotensive
Cerebral infarcts	0 (0%)	1 (5.0%)	16 (34.0%)	Eclampsia vs preeclampsia 0.29 (0.06–0.52) P=.012
Vasogenic cerebral edema	0 (0%)	4 (20.0%)	39 (80.0%)	Eclampsia vs normotensive 0.34 (0.03–0.65) P=.030
Vasospasm ^a	0 (0%)	1 (5.6%)	8 (18.0%)	Preeclampsia vs normotensive 0.05 (–0.09 to 0.19) P=.47
				Eclampsia vs normotensive 0.80 (0.47–1.00) P<.001
				Preeclampsia vs eclampsia 0.20 (–0.06 to 0.46) P=.13
				Eclampsia vs normotensive 0.18 (–0.06 to 0.43) P=.14
				Preeclampsia vs eclampsia 0.06 (–0.09 to 0.20) P=.45

Data are presented as number (percentage). Comparisons between groups were performed using the Farrington-Manning test for the RD.

CI, confidence interval; RD, risk difference.

^a Data on vasospasm are missing for 2 women in the preeclampsia group and 5 women in the eclampsia group. Bergman. *Pathophysiology of eclampsia*. *Am J Obstet Gynecol* 2025.

Magnetic resonance imaging findings in women with complex eclampsia

Women with only 1 eclamptic seizure and no other end-organ involvement were compared with women with complex eclampsia (multiple seizures and/or additional neurologic symptoms and/or other organ complications). There was no significant difference in the prevalence of cerebral infarcts (6/20 [30%] vs 10/29 [37%], respectively; $P=.61$) or vasogenic cerebral edema (15/20 [75%] vs 24/29 [83%], respectively; $P=.51$) between the groups. In addition, there was no difference in the prevalence of vasospasm (4/29 [15%] vs 4/20 [22%], respectively; $P=.56$).

There was no difference in diffusion or perfusion between women with complex eclampsia and those with eclampsia without additional complications (Supplemental Table 3).

Comment

Principal findings

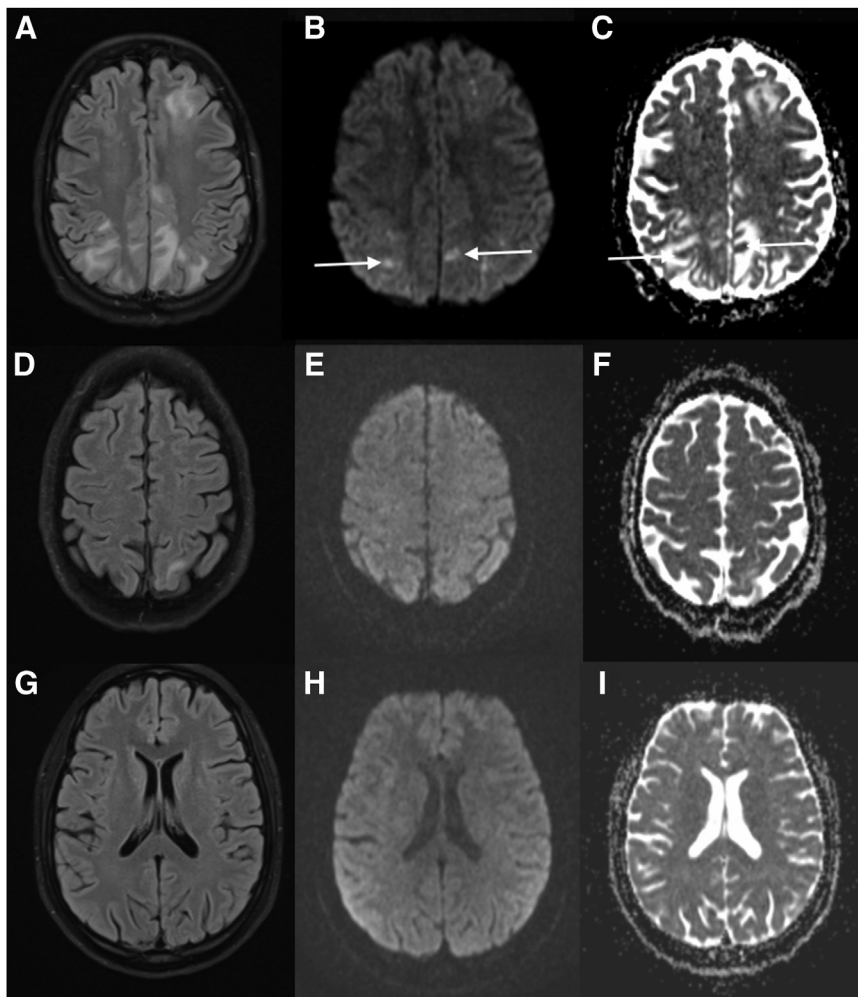
Cerebral infarcts and edema are common in women who experience eclampsia. Increased diffusion (subclinical edema) was observed in several regions in women with eclampsia compared with normotensive women and women with preeclampsia. Hyperperfusion was not observed in eclampsia and preeclampsia. In contrast, certain areas showed decreased cerebral capillary blood flow, especially when associated with cerebral edema. Vasospasm was present in 1 of 5 women with eclampsia.

Preeclampsia with severe features does not have similar pathology as eclampsia, and cerebral infarcts and vasospasm are uncommon. Of note, 1 of 5 women with severe preeclampsia had cerebral edema.

Results in the context of what is known

Previously, it was thought that eclampsia was reversible and not associated with long-term complications. We and others have shown that cerebral infarcts are common in eclampsia, which might be potentially irreversible.^{2,3,12} Interestingly, women who experience only 1 seizure without other end-organ

FIGURE 2
Cerebral edema and infarcts in eclampsia and preeclampsia

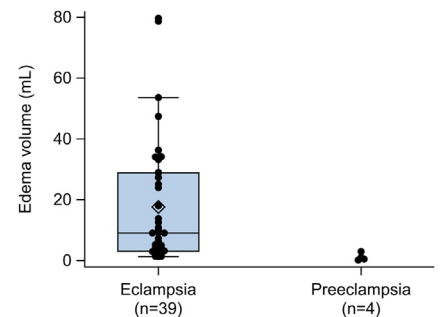


Magnetic resonance imaging demonstrating subcortical vasogenic cerebral edema in bilateral parietal lobes (FLAIR sequence [A]), with scattered small areas of ischemia (arrows) (diffusion-weighted sequence [B] and ADC map [C]) in a woman who experienced eclampsia. A smaller area of subcortical edema in a woman with preeclampsia (without eclampsia) is observed in the left parietal lobe (FLAIR image [D]), without ischemic lesions (diffusion-weighted sequence [E] and ADC map [F]). Corresponding FLAIR (G) and diffusion-weighted image (H) and ADC map (I) from a normotensive control show normal findings.

ADC, apparent diffusion coefficient; FLAIR, fluid attenuation inversion recovery.

Bergman. Pathophysiology of eclampsia. *Am J Obstet Gynecol* 2025.

FIGURE 3
Scatterplot of edema volume in eclampsia and preeclampsia



Points are observed values. The box limits are the lower and upper quartiles. The line within the box represents the median, and the diamond represents the mean. The box plot for the preeclampsia group is not shown because of a small sample size.

Bergman. Pathophysiology of eclampsia. *Am J Obstet Gynecol* 2025.

hyperperfusion, particularly in areas with cerebral edema. Therefore, it is unlikely that forced cerebral capillary vessel dilatation and hyperperfusion play a role in the development of eclampsia. The caudate nucleus is particularly affected by hypoperfusion in this South African cohort. This is following our previous results showing hypoperfusion of the caudate nucleus in women with preeclampsia compared with normotensive controls in a Swedish cohort.¹³

Vasospasm has been studied in some older case reports or series of women with eclampsia and preeclampsia.^{14–16} In some studies, vasospasm was found in several women with eclampsia and preeclampsia and resolved with a resolution of cerebral edema. In other studies, vasospasm was not present.^{17,18} These discrepancies may be due to differences in imaging technique and criteria for vasospasm and timing of imaging, as this is likely a dynamic process. Our results from the largest prospectively included cohort of women with eclampsia and preeclampsia support the presence of vasospasm in eclampsia.

involvement have a similar risk of cerebral infarcts to those who have experienced multiple seizures or eclampsia with other end-organ involvement. Imaging and ongoing neurologic follow-up may be warranted for all women who have experienced eclampsia.

Cerebral edema is common in eclampsia, affecting 80% of women in this cohort. In addition, cerebral

diffusion (subclinical cerebral edema) was increased in women with eclampsia, likely reflecting an increase in microscopic tissue water content in this group. Our study evaluated the diffusion in women with eclampsia. The result could be important for understanding the pathophysiology of eclampsia.

Our data show that eclampsia is associated with hyperperfusion and not

TABLE 3
Diffusion and perfusion in normotensive women and in women with preeclampsia or eclampsia

Variable	Normotensive (n=10)	Preeclampsia (n=20)	Eclampsia (n=49)	Mean difference (95% CI)		
				Eclampsia vs normotensive	Preeclampsia vs normotensive	Eclampsia vs preeclampsia
Diffusion (D, $\text{mm}^2/\text{s} \times 10^{-3}$)						
Frontal white matter	0.70 (0.06)	0.77 (0.11)	0.77 (0.08)	0.07 (0.02–0.12) ^a $P=.012^a$	0.07 (0.00–0.14) ^a $P=.048^a$	–0.00 (–0.06 to 0.06) $P=.95$
Parieto-occipital white matter	0.65 (0.03)	0.67 (0.03)	0.70 (0.06)	0.05 (0.02–0.07) ^a $P=.003^a$	0.02 (–0.01 to 0.05) $P=.12$	0.02 (0.00–0.05) ^a $P=.045^a$
Caudate nucleus	0.67 (0.04)	0.68 (0.03)	0.70 (0.04)	0.04 (0.00–0.07) ^a $P=.028^a$	0.02 (–0.01–0.05) $P=.25$	0.02 (0.00–0.04) ^a $P=.033^a$
Lentiform nucleus	0.67 (0.03)	0.68 (0.03)	0.69 (0.05)	0.02 (–0.01 to 0.04) $P=.15$	0.01 (–0.01 to 0.04) $P=.34$	0.01 (–0.01 to 0.03) $P=.47$
Thalamus	0.65 (0.02)	0.67 (0.03)	0.67 (0.02)	0.02 (–0.00 to 0.03) $P=.093$	0.01 (–0.01 to 0.03) $P=.33$	0.01 (–0.01 to 0.02) $P=.51$
Perfusion ($f \times D^*$, $\text{mm}^2/\text{s} \times 10^{-3}$)						
Frontal white matter	1.42 (0.44)	1.19 (0.37)	1.23 (0.33)	–0.19 (–0.51 to 0.14) $P=.23$	–0.22 (–0.57 to 0.12) $P=.19$	0.04 (–0.17 to 0.24) $P=.73$
Parieto-occipital white matter	1.23 (0.14)	1.29 (0.17)	1.23 (0.16)	–0.00 (–0.11 to 0.10) $P=.96$	0.06 (–0.06 to 0.18) $P=.33$	–0.06 (–0.16 to 0.03) $P=.19$
Caudate nucleus	1.11 (0.29)	1.32 (0.18)	1.15 (0.22)	0.04 (–0.18 to 0.25) $p=.72$	0.20 (–0.02 to 0.42) $p=.067$	–0.17 (–0.27 to –0.06) ^a $p=.003^a$
Lentiform nucleus	1.54 (0.35)	1.46 (0.21)	1.38 (0.21)	–0.16 (–0.42 to 0.10) $P=.19$	–0.08 (–0.34 to 0.18) $P=.52$	–0.08 (–0.20 to 0.04) $P=.18$
Thalamus	1.34 (0.17)	1.42 (0.11)	1.38 (0.16)	0.03 (–0.09 to 0.16) $P=.59$	0.07 (–0.05 to 0.20) $P=.23$	–0.04 (–0.11 to 0.03) $P=.23$

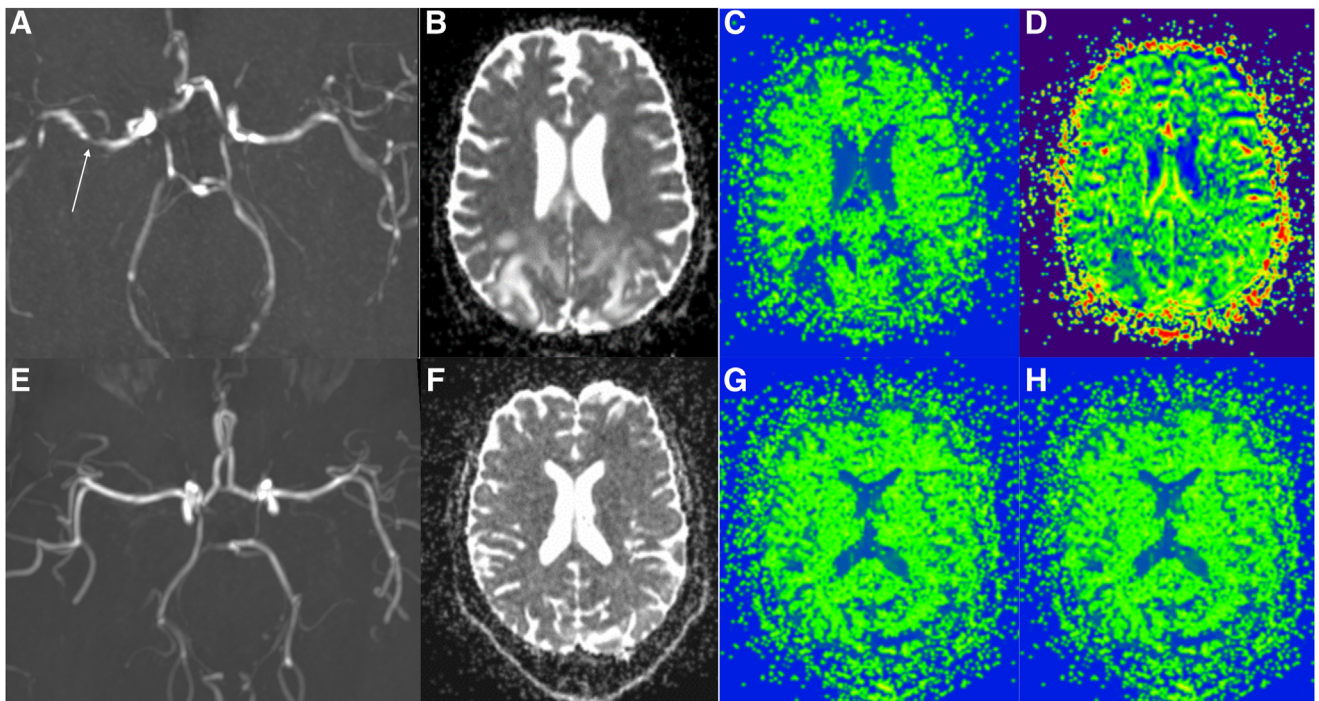
Data are presented as mean (standard deviation). Comparisons between groups were performed using the Welch t test, accounting for unequal variances between groups.

CI, confidence interval.

^a $P<.05$.

Bergman. Pathophysiology of eclampsia. *Am J Obstet Gynecol* 2025.

FIGURE 4
Vasospasm, diffusion, and perfusion in eclampsia and preeclampsia



MR images of cerebral arteries (A and E), diffusion (B and F), and perfusion (C, D, G, and H) in a woman with eclampsia (A–D) and a normotensive control (E–H). MR images from a woman with eclampsia show vasospasm (arrow in A), increased water diffusion (D in B), and low perfusion estimates (pseudo-diffusion, D^* in C, and perfusion fraction, f in D) in areas with edema.

MR, magnetic resonance.

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Research implications

Women with eclampsia who have cerebral infarcts may have an even higher risk of developing longer-term neurologic sequelae than those without infarcts and should be followed up in future research.

If cerebral perfusion is decreased in eclampsia, it is important to evaluate the effects of antihypertensive and neuroprotective treatments on

cerebral blood flow. The effect of acute decreases in systemic blood pressure on cerebral autoregulation and subsequent perfusion has not been studied.

Clinical implications

Conventional imaging does not rule out cerebral pathology in women who have experienced eclampsia and preeclampsia.

Women who have experienced preeclampsia and eclampsia may have an increased risk of neurologic long-term sequelae, such as epilepsy, cognitive decline, and dementia, in this population irrespective of normal conventional imaging.^{19–21} Long-term follow-up and education about the potential consequences of eclampsia may be needed.

Women presenting with neurologic signs and symptoms who undergo a

TABLE 4
Diffusion and perfusion in edema regions in women with preeclampsia compared with women with eclampsia

Variable	Eclampsia(n=32)	Preeclampsia(n=3)	Fold change (95% CI)	P value
Diffusion (D , $\text{mm}^2/\text{s} \times 10^{-3}$)	1.30 (1.20–1.60)	1.40 (1.20–1.40)	1.02 (0.81–1.26)	.81
Perfusion ($f \times D^*$, $\text{mm}^2/\text{s} \times 10^{-3}$)	0.17 (0.10–0.30)	0.36 (0.3–0.4)	0.57 (0.26–1.33)	.19

Data are presented as median (interquartile range). Data were missing for 1 woman in the preeclampsia group and 7 women in the eclampsia group. Comparison between groups was performed using nonparametric permutation test for the mean difference on log-transformed variables. Corresponding CIs were calculated using test inversion.

CI, confidence interval.

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TABLE 5

Diffusion and perfusion in edema regions vs parieto-occipital white matter without edema in 35 women with cerebral edema

Variable	Edema regions	Parieto-occipital white matter	Mean difference (95% CI)	P value
Diffusion (D , $\text{mm}^2/\text{s} \times 10^{-3}$)	1.38 (0.24)	0.70 (0.05)	0.68 (0.59–0.76)	<.001
Perfusion ($f \times D^*$, $\text{mm}^2/\text{s} \times 10^{-3}$)	0.25 (0.18)	1.25 (0.17)	–1.00 (–1.09 to –0.91)	<.001

Descriptive data are presented as mean (standard deviation). Data to calculate diffusion and perfusion in edema regions were missing for 1 woman in preeclampsia group and 7 women in the eclampsia group. Statistical analyses were performed using the paired *t* test.

CI, confidence interval.

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normal conventional brain MRI may still have preclinical brain edema and perfusion defects.

Strengths and Limitations

This was a study of women with eclampsia who underwent MRI using a protocol to assess diffusion, perfusion, and vasospasm. We present novel findings of preclinical edema, perfusion deficits, and vasospasm in eclampsia and preeclampsia. MRI examinations were performed in women who did not qualify for clinically indicated imaging in our setting. Women suspected of stroke were not included. The study was prospectively conducted, and MRI sequences were interpreted by blinded neuroradiologists.

This study has limitations. All imaging was performed after the onset of complications. Therefore, it is not possible to draw conclusions about factors present before the development of eclampsia. For the perfusion estimates, the lack of differences between groups could be due to the relatively low magnetic field and use of the IVIM technique for perfusion estimates, thereby decreasing the signal-to-noise ratio. However, an important advantage is that IVIM does not require contrast agent administration, which is why we chose to use it in this study. This allowed for antepartum measurements if needed and allowed imaging in cases of renal lesions where contrast agent administration for research would be ethically questioned. Thus, repeating the MRI investigation with higher field strength and/or contrast agent–based perfusion measurements could be of value.

Ideally, we would want to image women before an eclamptic seizure to evaluate whether the edema is present and precedes eclampsia. This is extremely challenging as eclampsia is unpredictable and MRI examinations are expensive and not readily available, particularly in settings where eclampsia is common.

Conclusions

Cerebral infarcts were present in one-third of women who experienced eclampsia, independent of disease severity. Women with preeclampsia and eclampsia exhibited preclinical cerebral edema (increased diffusion). This may explain neurologic signs and symptoms in these women when there is no clinically evident cerebral edema on conventional MRI. Hyperperfusion with forced capillary dilatation does not seem to be the underlying cause of edema in eclampsia. Vasospasm with decreased capillary blood flow (hypoperfusion) is more likely to contribute to cerebral edema formation and subsequent neuroinflammation in preeclampsia and eclampsia. A conventional MRI does not rule out pathology in preeclampsia and eclampsia. ■

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SUPPLEMENTAL TABLE 1
MRI sequence parameters

Sequence	Type	Orientation	Spatial resolution (mm)	TR (ms)/TE (ms)/flip angle (degree)	Number of excitations
T1 weighted	2D turbo spin echo	Sagittal	0.6 × 0.6 × 4.0	389.0/9.7/150.0	3
T2 weighted	2D turbo spin echo	Axial	0.6 × 0.6 × 4.0	5720/80/150	3
T2* weighted	2D gradient echo	Axial	0.4 × 0.4 × 4.0	830/25/20	1
FLAIR	2D IR-spin echo	Axial	0.7 × 0.7 × 4.0	8000/84/150	1
Diffusion weighted	2D echo planar imaging	Axial	1.25 × 1.25 × 3.00	6500/119/90	1
MR angiography	3D time-of-flight	Axial	0.4 × 0.4 × 0.5	27/7/25	1

MRI was performed on 1.5T (Aera; Siemens Healthineers, Erlangen, Germany). The examination protocol included sagittal 2D T1-weighted spin echo, axial 2D T2 spin echo, axial 2D T2 gradient echo, axial 2D FLAIR, and axial DWI. The DWI sequence was acquired with 9 b values (0, 50, 100, 150, 200, 400, 600, 800, and 1000 s/mm² × 10⁻³) for separation of diffusion and perfusion parameters. The assessment of edema, ischemia, diffusion, and perfusion were performed by a blinded radiologist (D.H.). Vasogenic edema was evaluated on FLAIR images and quantified using manual outlining on individual slices. Edema volume was calculated as the sum of these areas multiplied by slice thickness. The presence of ischemic lesions was assessed using the b₁₀₀₀ DWI images, where high signal intensity corresponding to cytotoxic edema was interpreted as a sign of acute ischemia. The intravoxel incoherent imaging parametric maps (D, D*, and f) were calculated from the multiple b-value DWI sequence using commercially available software (Olea Sphere 3.0, Olea Medical, La Ciotat, France). These metrics correspond to the degree of water self-diffusion (D), pseudo-diffusion related to capillary blood flow (D*), and volume fraction of perfused capillaries (f) as described by Le Bihan et al.⁵ D, D*, and f were measured in the following regions: frontal white matter, parieto-occipital white matter, caudate nucleus, lentiform nucleus, and thalamus. Estimates were obtained from averages of the right and left sides in 2 or 3 slices.

2D, 2-dimensional; 3D, 3-dimensional; DWI, diffusion-weighted imaging; FLAIR, fluid attenuation inversion recovery; IR, inversion recovery; MR, magnetic resonance; MRI, magnetic resonance imaging; TE, time to echo; TR, repetition time.

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SUPPLEMENTAL TABLE 2

Characteristics of women with eclampsia with and without MRI in the database

Variable	With MRI (n=49)	Without MRI (n=52)
Demographics and baseline characteristics		
Age (y), mean (SD)	22.4 (5.6)	22.6 (6.3)
Body mass index (kg/m ²), mean (SD)	24.8 (5.6)	28.6 (7.6)
Nulliparous	36 (73.5%)	32 (61.5%)
Any antenatal care	41 (83.7%)	42 (80.4%)
HIV positive	6 (12.2%)	6 (11.5%)
Smoking during pregnancy	9 (18.4%)	4 (7.7%)
Alcohol use during pregnancy	7 (14.3%)	2 (3.8%)
Gestational diabetes mellitus	0 (0%)	1 (1.9%)
Chronic hypertension	2 (4.2%)	3 (5.8%)
Mode of birth		
Vaginal	17 (34.7%)	12 (23.1%)
Elective or nonurgent CD	1 (2.0%)	0 (0%)
Emergency CD	31 (63.3%)	39 (75.0%)
Gestation at delivery (wk), median (IQR)	34 4/7 (24 2/7 to 40 5/7)	34 2/7 (23 0/7 to 41 6/7)
Live-born infant	41 (83.7%)	42 (82.4%)
Birthweight (g), mean (SD)	2076 (946)	2125 (914)
Maternal complications		
Maternal death	0 (0%)	3 (5.8%)
Intensive care unit admission	4 (8.2%)	6 (11.5%)
Recurrent eclampsia	17 (34.7%)	17 (32.7%)
Glasgow Coma Scale of <13	12 (24.5%)	9 (17.3%)
Cortical blindness	2 (4.1%)	3 (5.8%)
Pulmonary edema	1 (2.0%)	3 (5.8%)
Inotropic support	1 (2.0%)	2 (3.8%)
Renal impairment	10 (20.4%)	4 (7.7%)
Dialysis	1 (2.0%)	0 (0%)
HELLP syndrome	14 (28.6%)	10 (19.2%)
DIC INR of >1.2	10 (20.4%)	1 (1.9%)
Severe hypertension	17 (34.7%)	20 (38.5%)
Sepsis	4 (8.2%)	5 (9.6%)
Venous thromboembolism	1 (2.0%)	1 (1.9%)
Placental abruption	3 (6.1%)	2 (3.8%)

Data are presented as mean (SD) or median (IQR) for numeric variables and number (percentage) for categorical variables.

CD, cesarean delivery; DIC, disseminated intravascular coagulation; HELLP, hemolysis, elevated liver enzymes, and low platelet count; INR, international normalized ratio; IQR, interquartile range; MRI, magnetic resonance imaging; SD, standard deviation.

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SUPPLEMENTAL TABLE 3

Magnetic resonance imaging findings in women with eclampsia or eclampsia with complications

Variable	Eclampsia with complications (n=29)	Eclampsia (n=20)	Mean difference (95% CI)	P value
Diffusion (D , $\text{mm}^2/\text{s} \times 10^{-3}$)				
Frontal white matter	0.78 (0.10)	0.76 (0.06)	0.02 (−0.03 to 0.07)	.46
Parieto-occipital white matter	0.70 (0.06)	0.70 (0.05)	0.01 (−0.03 to 0.04)	.74
Caudate nucleus	0.71 (0.04)	0.69 (0.03)	0.02 (−0.01 to 0.04)	.19
Lentiform nucleus	0.69 (0.06)	0.68 (0.04)	0.01 (−0.02 to 0.04)	.36
Thalamus	0.67 (0.03)	0.67 (0.02)	−0.00 (−0.02 to 0.01)	.68
Perfusion ($f \times D^*$, $\text{mm}^2/\text{s} \times 10^{-3}$)				
Frontal white matter	1.21 (0.28)	1.25 (0.40)	−0.05 (−0.26 to 0.17)	.67
Parieto-occipital white matter	1.24 (0.16)	1.21 (0.18)	0.03 (−0.07 to 0.13)	.56
Caudate nucleus	1.10 (0.24)	1.21 (0.19)	−0.11 (−0.24 to 0.02)	.097
Lentiform nucleus	1.37 (0.20)	1.39 (0.23)	−0.03 (−0.16 to 0.10)	.68
Thalamus	1.36 (0.19)	1.40 (0.11)	−0.04 (−0.13 to 0.05)	.38

Data are presented as mean (standard deviation). Statistical analyses were performed using the Welch *t* test.

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