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Cranial MR imaging with clinical correlation in preeclampsia and eclampsia

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PURPOSE
Our aim was to determine the distribution and nature of cranial MRI findings in preeclampsia/eclampsia, and also to correlate them with clinical and laboratory data.

MATERIALS AND METHODS
MR imaging was performed in 39 patients with preeclampsia (n=30) and eclampsia (n=9), and the distribution and signal patterns of the lesions were documented. Clinical findings, blood pressures, and laboratory data were compared statistically in patients with and without MR imaging findings.

RESULTS: MR imaging was normal in 21 of the patients. In 18 patients, cortical-subcortical lesions, which appeared iso-/hypointense on T1W and hyperintense on T2W images, were detected. The occipital lobe was involved in all patients, followed by the parietal, frontal, and temporal lobes, and basal ganglia and pons. The lesions showed watershed distribution in 13 patients. When the patients with and without MR imaging findings were compared, there was a statistically significant difference regarding visual disturbances, depression of consciousness, and seizures (p=0.042, p=0.006, p=0.000, respectively). Although patients with MR imaging findings showed higher blood pressures as compared to those without MR imaging findings, there was no statistically significant difference (p=0.074). In patients with MR imaging findings, lactate dehydrogenase (LDH), uric acid, and creatinine levels were significantly higher than those without MR imaging findings (p=0.006, p=0.010, p=0.005, respectively).

CONCLUSION: Increased permeability of the blood-brain-barrier related to endothelial injury plays a major role in the pathogenesis of preeclampsia/eclampsia. Relatively minor increases in blood pressure may cause cerebral lesions. However, when the cerebral autoregulation mechanism is considered, the distribution of cerebral lesions in the posterior circulation and watershed zones, which are relatively sparsely innervated by sympathetic nerves, provides evidence that the main determinant of pathogenesis is acute fluctuations in blood pressure.

Key words: ● *preeclampsia* ● *eclampsia* ● *magnetic resonance imaging*

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Preeclampsia is a disease characterized by hypertension, peripheral edema, and proteinuria, which affects 4%-5% of all pregnancies. Many of the patients with preeclampsia present with headache, visual disorders, confusion, and depression of consciousness. The disease is referred to as eclampsia when seizure is a component (1).

Pathological conditions, which present with headache, visual disorders, seizure, and depression of consciousness, and that regress both clinically and neuroradiologically in a few weeks with the elimination of etiological factors have different names such as "posterior reversible encephalopathy syndrome" (PRES), "hypertensive encephalopathy", "reversible posterior cerebral edema syndrome" and "posterior reversible leukoencephalopathy syndrome" (PRLS). Recently, the term PRES (3) has become preferable to the term PRLS (2), which was first described in 1996, because of the gray matter involvement accompanying white matter involvement in these patients. PRES can occur in conditions such as hypertension, preeclampsia/eclampsia, immunosuppressive treatment (cyclosporin A, tacrolimus), and uremia (1, 2, 4-7). Vasogenic edema, most prominently at the posterior regions of the cerebral hemispheres, and less commonly, the brain stem, cerebellum, and basal ganglia, is detected on computed tomography (CT) and magnetic resonance (MR) imaging examinations (1, 2, 4, 5, 7-9). Cytotoxic edema and irreversible brain damage occur if etiological factors remain or are not treated (10-12).

The cause of preeclampsia/eclampsia, which threatens the lives of mother and fetus, has not yet been explained. Investigation of the clinical parameters associated with brain lesions detected in preeclampsia/eclampsia cases might shed light to the pathogenesis of the disease (1, 2, 13). Schwartz et al. demonstrated that brain lesions detected in MR examination are associated with endothelial damage indicators, not hypertension (1). The purpose of this study was to determine the distribution and nature of cranial MR imaging findings in preeclampsia/eclampsia, and also to correlate them with clinical and laboratory data.

Materials and methods

A total of 39 patients diagnosed with preeclampsia (n=30) and eclampsia (n=9) between 2000-2003 composed the study group. Ten of the patients were evaluated retrospectively and 29 of them prospectively. Age range was 18-39 years (mean, 26.2 years). Two of the patients were diagnosed with HELLP syndrome (hemolysis, elevated liver enzymes, and thrombocytopenia). Two of the patients were admitted to the emergency department with premature rupture of membranes and were diagnosed with preeclampsia. Three of the patients had seizures in the postpartum period. All of the patients were discharged from the hospital following delivery and without sequela.

Age, systolic and diastolic arterial blood pressure, mean arterial blood pressure, hematocrite (Hct), mean eritrocyte volume (MEV), white blood cell count (WBC), thrombocyte, lactate dehydrogenase (LDH), aspartate transaminase (AST), alanine transaminase (ALT), urea, uric acid, albumin, globulin, and calcium (Ca) values of all patients were recorded. Mean arterial tension values were calculated with 1/3x (systolic pressure + 2 x diastolic pressure) formula. Cranial MR examinations were performed for all patients between the 1st and 7th day of the onset of clinical symptoms in a 0.5T magnetic resonance imaging system. The examination protocol consisted of T1-weighted spin echo (axial and sagittal) and T2-weighted spin echo axial sequences without intravenous contrast material injection. Control MR examinations were performed for two of the patients two months after the first examination.

Clinical and laboratory data of all patients, with or without pathological findings on cranial MR imaging, were compared statistically. As HELLP syndrome is associated with fulminant hematological anomalies (14) and can erroneously affect laboratory results, MR findings of the two HELLP cases were excluded from the statistical analyses of laboratory values. All statistical analyses were performed with the SPSS 11.0 program for Windows. Mann-Whitney U test and chi-square test were used for statistical evaluation and $p<0.05$ was accepted to be statistically significant and $p\geq0.005$ was accepted to be statistically insignificant. Data dependent upon verbal explanations were depicted as frequency and %, data dependent upon laboratory parameters were depicted as mean ±SD.

Table 1. Clinical findings and distribution of lesions in patients with MR imaging findings

Patient No.	Occipital	Parietal	Frontal	Temporal	Basal ganglia	Pons	Clinical findings
1	+	+	+	+	+	-	HA, S, DC
2	+	+	+	+	+	+	HA, S, DC
3	+	+	-	-	+	-	HA, VD
4	+	+	-	+	-	-	HA, VD
5	+	+	-	+	-	-	HA, VD, S, DC
6	+	+	+	-	+	-	HA, VD
7	+	-	-	-	+	-	HA, DC
8	+	+	+	+	+	-	HA, VD, DC
9	+	-	-	-	-	-	HA
10	+	+	-	-	-	-	HA, VD
11	+	-	-	-	-	-	HA
12	+	+	+	+	+	-	HA, S, DC
13	+	+	+	+	+	-	HA, S
14	+	+	+	+	+	-	HA, VD, S
15	+	+	-	-	-	-	HA, VD
16	+	+	+	+	+	+	HA, S, DC
17	+	-	-	-	-	-	HA, VD, S
18	+	+	-	-	-	-	HA, VD, S, DC
Total	100%	77.7%	50%	50%	55.5%	11.1%	

HA: headache, S: seizure, DC: depression of consciousness, VD: visual disorder

Table 2. Clinical findings in patients with and without positive MR imaging findings, and their statistical comparison

	MR imaging positive (n=18)	MR imaging negative (n=21)	p
Headache	18	19	0.490
Visual disorder	10	4	0.042*
Depression of consciousness	8	1	0.006*
Seizure	9	0	0.000*

*Statistically significant p values with chi-square test

Results

Of our 39 patients, 37 (94.9%) had headaches, 14 (35.9%) had visual disorders, 9 (23%) had seizures, and 9 (23%) had depression of consciousness. Cranial MR imaging was found to be normal in 21 patients (53.8%). In the remaining 18 patients, cortical-subcortical lesions that were iso-/hypointense on T1-weighted images and hyperintense on T2-weighted images were detected. Subcortical white matter was affected in only 8 of these 18 patients (44.4%) and pathological signals were present in both the cortex and the white matter in the remaining 10 patients (55.6%). Anterior, posterior, or parasagittal watershed zone involvement was present in 13 patients (72.2%). Clinical findings and distribution of lesions in patients with MR findings are presented in Table 1. Pathological findings regressed in two patients who had control examinations about two months after the first examination (Figures 1-3).

Clinical findings and statistical analyses of patients with or without MR findings are shown in Table 2. When the patients with or without MR imaging findings were compared, there was a statistically significant difference regarding visual disturbances, depression of consciousness, and seizures, but there was no significant difference regarding headaches. There was no statistically significant difference between the mean age of patients with or without MR imaging findings (Mann-Whitney U test, p=0.097). Although patients with MR imaging findings showed higher blood pressures as compared to those without MR findings, there was no statistically significant difference between the two groups (Mann-Whitney U test, p=0.074). In Table 3, minimum and maximum mean blood

pressure values, as well as their means, and statistical comparison of the patient groups with or without MR findings are given. Biochemical data, lactate dehydrogenase (LDH), uric acid, and creatinine levels were significantly higher in patients with positive MR findings than those without MR findings (Mann-Whitney U test, p=0.006, p=0.010, p=0.005, respectively).

Discussion

Pathogenesis in PRES patients is not yet fully understood, but a major causative factor is thought to be the extravasation of fluid (15). Brain lesions detected in these patients might be related to a disturbance of the cerebral autoregulation mechanism and impairment of endothelial function (1, 8, 16). The cerebral autoregulation mechanism, consisting of myogenic and neurogenic components, maintains stable blood perfusion in normal individuals (1). Effective functioning of neurogenic mechanisms depends on sympathetic innervation. In PRES cases, direct toxic effects on endothelium or vessel distention, which depends on elevated blood pressure, decreases the effect of myogenic mechanisms (1, 4). In these cases, neurogenic mechanisms take over the regulation of cerebral perfusion; this way, posterior circulation areas, which are relatively sparsely innervated by sympathetic nerves, become more sensitive to blood pressure elevations. In PRES cases with hypertension, serum extravasation occurs when the elevation in blood pressure passes beyond the autoregulation capacity of brain blood vessels. Brain lesions are more commonly demonstrated in posterior areas in these cases. Vasoconstriction, which sympathetic innervation induces, moderately protects anterior circulation areas from overperfusion. In PRES cases without hypertension, direct endothelial cell dysfunction, which increases blood-brain barrier permeability, is thought to be responsible for the pathogenesis (1, 8).

In this study, various clinical and laboratory parameters in preeclampsia/eclampsia cases with and without positive brain MR imaging findings were compared. Brain lesions were found to be associated with high LDH levels, which is an indicator of hemolysis, and both high uric acid and creatinine levels, which are indicators of renal function disorder. Endothelial injury yields to morphological disturbances in erythrocytes and microvascular hemolysis; as a result, LDH level increases (1). The cause of endothelial injury in preeclampsia/eclampsia cases has not yet been demonstrated, but circulating endothelial toxins or endothelium antibodies are thought to be responsible (1). In previous studies, it has been demonstrated that in preeclampsia/eclampsia patients, high LDH levels occur before lesions appear in brain MR examination, and this finding shows that high blood pressure does not lead to endothelial injury (1, 17). Multiple organ involvement in preeclampsia/eclampsia cases is thought to be associated with an endothelial function disorder (17). Renal function disorder secondary to renal endothelial injury results in uric acid and creatinine increase.

In our study, there was no statistically significant difference between blood pressure values of cases with or without MR imaging evidence of brain lesions. But in cases of preeclampsia/eclampsia, brain lesions might occur although blood pressure values are normal but still higher than a patient's routine normal blood pressure (1). When blood pressure values and results associated with laboratory parameters were evaluated together in our study, brain edema detected in preeclampsia/eclampsia was thought to be secondary to endothelial injury, rather than hypertension. These findings correlated with the findings of Schwartz et al. (1). But the distribution of brain lesions should also be evaluated in determining whether hypertension or endothelial injury is more responsible for the pathogenesis.

Table 3. Comparison of biochemical data and mean blood pressure in patients without and with positive MR imaging findings

	MR imaging negative			MR imaging positive			p
	Minimum	Maximum	Mean±SD	Minimum	Maximum	Mean±SD	
Hct (%)	24.40	45.00	33.45±6.13	28.00	45.40	34.59±4.96	0.469
WBC (10^3/µl)	9.0	24.00	13.49±3.22	7.30	18.40	12.79±3.25	0.748
Thrombocyte (10^3/µl)	86.00	515.00	264.71±104.88	96.00	356.00	245.20±70.03	0.432
MEV	62.40	96.90	86.76±7.33	78.10	93.80	89.76±4.25	0.123
LDH (U/l)	322	925	605.48±184.51	501	1012	792.40±186.08	0.006*
AST (U/l)	15	75	35.76±15.67	20	39	31.93±6.78	0.797
ALT (U/l)	6	93	26.24±18.89	7	39	25.60±8.67	0.712
Uric acid (mg/dl)	2.80	10	4.41±1.83	3.10	7.30	5.61±1.17	0.010*
Urea (mg/dl)	12.9	128	30.45±24.26	6	58.0	33.40±12.31	0.077
Albumine (g/dl)	2.20	4.0	2.99±0.55	2	4	2.87±0.57	0.584
Globuline (g/dl)	1.80	3.10	2.66±0.35	2.0	3.20	2.60±0.36	0.438
Ca (mEq/dl)	5.70	8.0	7.19±0.68	5.60	7.90	7.26±0.69	0.675
Creatinine (mg/dl)	0.50	1.10	0.68±0.14	0.6	2.10	0.86±0.31	0.005*
Mean BP (mmHg)	100	146	123.81±11.70	116	166	131.87±13.41	0.074

SD: standard deviation, Hct: hematocrit, MEV: mean erythrocyte volume, WBC: white blood cell count, LDH: lactate dehydrogenase, AST: aspartate transaminase, ALT: alanine transaminase, Ca: calcium, BP: blood pressure.

*Statistically significant p values with Mann-Whitney U test

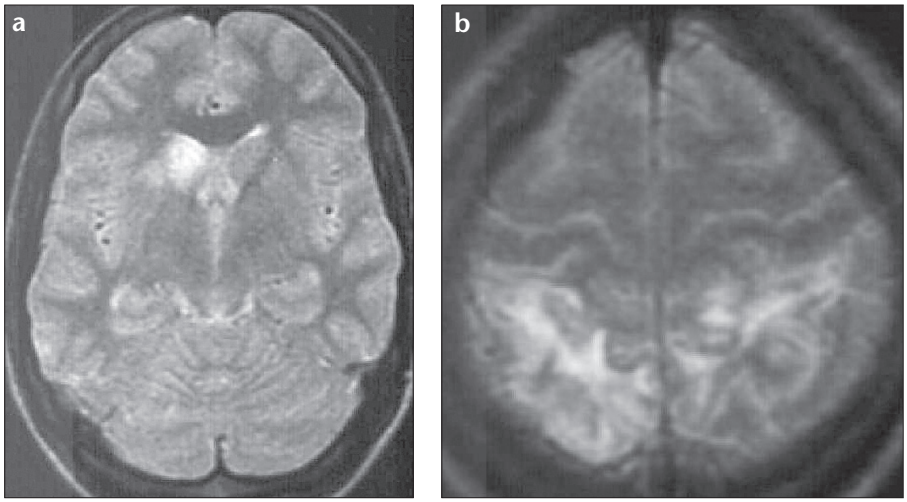

Figure 1. a, b. Eclampsia. A 23-year-old patient, 36 weeks pregnant, with headache for one week has been hospitalized for new onset visual loss and generalized tonic clonic convulsion. Axial T2-weighted MR image (**a**) shows high-signal lesion in the right head of caudate and partially the lentiform nucleus. Axial T2-weighted image at a superior level (**b**) shows bilateral parietooccipital high-signal lesions involving the cortex and the white matter.

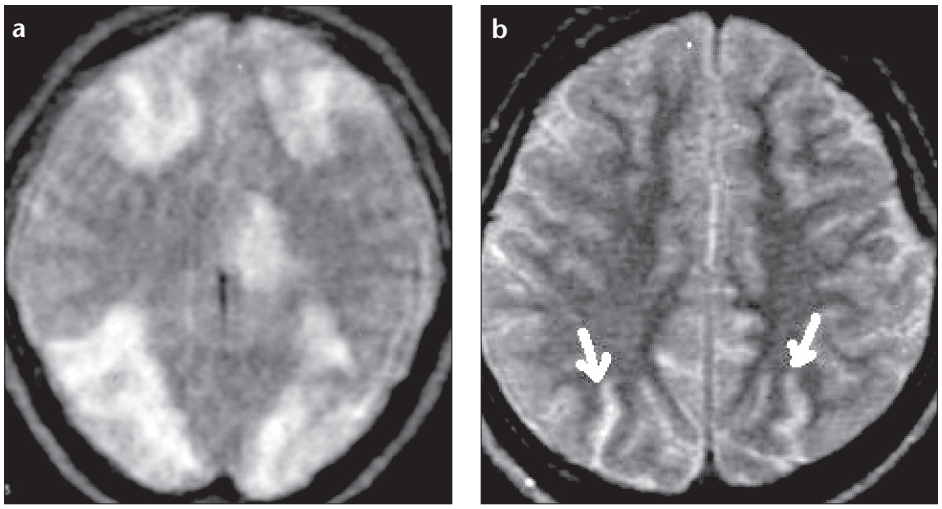

Figure 2. a, b. Preeclampsia; cranial MR imaging of a 19-year-old patient, 38 weeks pregnant, with developing headache, visual loss, and depression of consciousness. Axial T2-weighted MR image (**a**) shows bilateral frontoparietal and occipital lesions involving the cortex and the white matter. Two months later (**b**) the lesions have disappeared except for minimal bilateral parietal subcortical hyperintensities *(arrows)*.

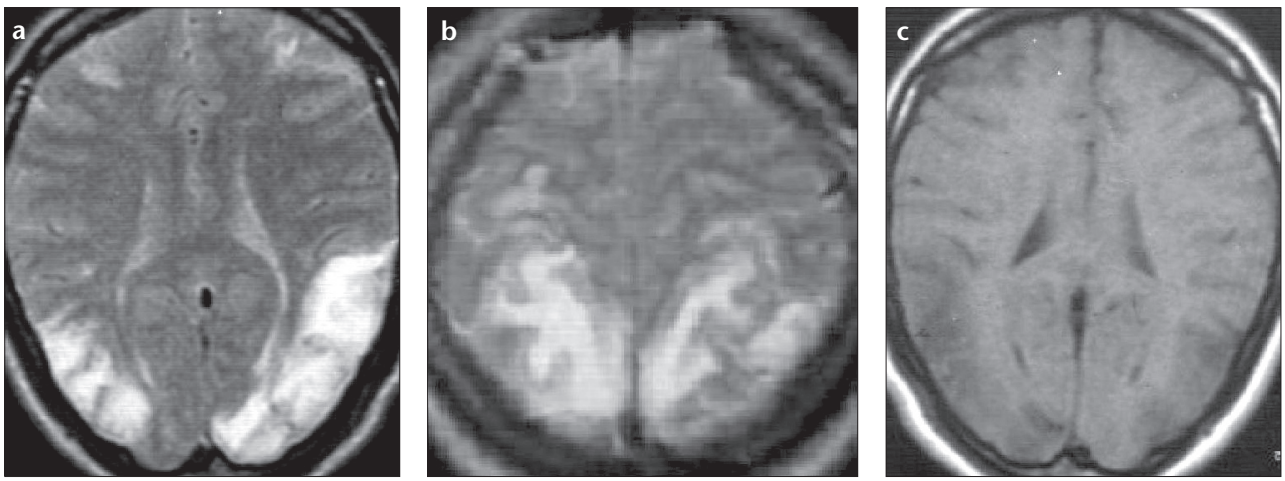

Figure 3. a-c. Preeclampsia. Cranial MR imaging of a patient, 34 weeks pregnant, with headache for a few weeks and developing acute visual loss. Axial T2-weighted MR images **(a** and **b)** show hyperintensities in bilateral frontal and parietooccipital lobes, quite symmetrical, involving anterior and posterior watershed zones cortically and subcortically. Axial T1-weighted MR image **(c)** shows the lesions to be iso-/hypointense.

We considered that PRES cases might be evaluated in three groups according to pathogenesis. In the first group, brain edema develops only secondary to elevated blood pressure. In these cases, lesions occur only in cases of high blood pressure values, as there is no endothelial injury leading to fluid extravasation. Limits of cerebral autoregulation pass beyond the normal levels of blood pressure and vasogenic edema occurs in posterior circulation areas, especially in the occipital lobes and watershed zones, which are relatively sparsely innervated by sympa-

thetic nerves. Arterial watershed zones have been demonstrated not be rich in sympathetic innervation as are posterior circulation areas (18). Cases of hypertensive encephalopathy occur in this group. Distribution of lesions in the second group is similar to the first group, but lesions might occur in cases of lower blood pressure values, even those close to normal. Endothelial injury plays a major role in the pathogenesis of these cases; as a result of a failure of the blood-brain barrier, fluid might leak into the interstitium in milder blood pressure elevations. As lesions occur especially in areas of low autoregulation capacity, acute blood pressure fluctuations are thought to be a major determinant in pathogenesis. In experimental studies, acute blood pressure elevations were shown to impair the autoregulation mechanism and cause fluid and blood leakage as a result of vasoconstriction and vasodilatation, especially in arterial watershed zones (19). Preeclampsia/eclampsia, cyclosporin-A, and tacrolimus toxicity are placed in this group (1, 5, 20, 21). In the third group, lesions predominantly occur secondary to endothelial injury; blood pressure elevations do not play a major role in pathogenesis. Symmetrical basal ganglion involvement is typical in these cases; thalamus, cerebellum, and brain stem involvement follow this. Occipital lobe and watershed zone involvement is not as prominent as they are in PRES cases with hypertension. Some kinds of uremic encephalopathy, especially hemolytic uremic syndrome, are placed in this group. (22). In uremic pathologies with renal hypertension, lesion distribution might be similar to the first two groups (23). By evaluating blood pressure measurements and the distribution of brain lesions, an understanding of pathogenesis might be possible in PRES cases with the classifications we suggest.

Lesions are generally iso-/hypointense on T1-weighted images, hyperintense on T2-weighted images, and petechial hemorrhage is rarely seen in patients with preeclampsia/eclampsia (1, 13, 24). Lesions with similar signal changes were demonstrated in all of our cases, but no findings of hemorrhage were detected. The occipital lobe is the most affected region in preeclampsia/eclampsia; parietal, frontal, temporal lobe, and basal ganglion involvement follow this. The cerebellum and brain stem might be involved in more severe cases (1, 24). The occipital lobe was involved in all of our cases. Lobar distribution of lesions was similar to other series in the literature, and watershed zone involvement was prominent in our cases. Watershed zone-distributed lesions evocate ischemia secondary to cerebral vasospasm, but as they are demonstrated not to be cytotoxic edema with diffusion weighted images, the possibility of ischemia is not an issue of concern (4).

If radiological and clinical findings occurring in PRES are easily recognized and treated immediately, they might be totally reversible (25). All the patients investigated in our study were discharged from the hospital without neurological deficit within 1-2 weeks. Control brain MR examination was performed in two of our patients two months after the first examination. Total recovery was detected in one of the patients and subcortical hyperintense lesions were present in a very limited area in the other patient. Ischemia, massive infarct, and death might occur if the disease is not recognized. Diffusion weighted MR imaging is used to distinguish ischemia-cytotoxic edema from vasogenic edema, which is present in PRES cases (4, 11, 12, 16). Cases that develop cytotoxic edema-ischemia as a complication of PRES have been reported (11, 12). It is accepted that massive vasogenic edema might cause an increase in tissue pressure and ischemia by impairing microcirculation (11).

Visual disorders, depression of consciousness, and seizures were more frequently detected in cases of preeclampsia/eclampsia with cerebral edema in MR imaging than in patients with normal MR examinations. Brain lesions were found to be significantly correlated with only seizure in the study of Schwartz et al. (4). This diversity might be explained by the widespread distribution of brain edema areas in our study. Seizure is thought to be secondary to the irritative effect of fluid in the cortex and white matter (1). Cerebral edema was present in our all cases with seizures. Depression of consciousness was demonstrated to be more frequent in cases with basal ganglion involvement in some studies (26). In 6 of the 10 (60%) patients with depression of consciousness in our study, basal ganglion involvement was present. There was no statistically significant difference between patients with or without MR imaging findings regarding headaches. The detection of visual disorders, depression of consciousness, or seizure in the follow-up of pregnant patients with preeclampsia/eclampsia should be a warning for possible brain lesions.

In conclusion, increased permeability of the blood-brain barrier related to endothelial injury plays a major role in the pathogenesis of preeclampsia/eclampsia. In cases where blood pressure elevations do not reach very high levels, intravascular fluid leaks into interstitium and vasogenic edema occurs. However, when the cerebral autoregulation mechanism is considered, the distribution of cerebral lesions in the posterior circulation and watershed zones, which are relatively sparsely innervated by sympathetic nerves, there is evidence that the main determinant of pathogenesis is acute fluctuations in blood pressure. When symptoms like visual disorders, depression of consciousness, or seizure develop during the follow-up of pregnancy, or indicators of endothelial injury are detected to be elevated, appropriate precautions to regulate blood pressure would be beneficial.

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