İSTANBUL UNIVERSITY PRESS

ARAŞTIRMA MAKALESİ / RESEARCH ARTICLE
Journal of Advanced Research in Health Sciences / Sağlık Bilimlerinde İleri Araştırmalar Dergisi 2023
DOI: 10.26650/JARHS2023-1226562

IS WHAT WE KNOW ABOUT POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME (PRES) TRUE?

POSTERİOR REVERSİBL ENSEFALOPATİ SENDROMU (PRES) HAKKINDA BİLDİKLERİMİZ DOĞRU MU?

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Citation/Atıf: Karabay Akgul O, Okuyan E, Kovalak EE, Akinci GE, Sevdi MS, Guraslan H, et al. Is what we know about posterior reversible encephalopathy syndrome (PRES) true? Journal of Advanced Research in Health Sciences 2023;6(2):143-148. https://doi.org/10.26650/JARHS2023-1226562

ABSTRACT

Objective: The aim of this study is to evaluate the risk factors and clinical course in cases of posterior reversible encephalopathy syndrome (PRES).
Material and Methods: In this study, we retrospectively reviewed the data of pregnant or puerperal women diagnosed with PRES in the tertiary center emergency obstetrics outpatient clinic and intensive care unit between 2017 and 2022. All patients were evaluated by obstetrics, neurology, ophthalmology, radiology, and intensive care physicians, and blood tests and imaging were performed in the same center. Application complaints, laboratory values, imaging methods, comorbidities, mode of delivery, and postpartum period were evaluated for each patient.
Results: In five years, a total of seven patients were diagnosed with PRES based on imaging methods and clinical findings. One of these patients had PRES twice, three years apart. Six of them presented with eclampsia. One patient was diagnosed with PRES postpartum in the first week, while other patients were diagnosed at pregnancy. Four patients had blurred vision, two patients had blindness, and one patient had no visual complaints. Three of the patients had mood changes (one patient confused, two patients agitated). One of the patients had diabetes mellitus (DM), which was known and treated with oral antidiabetics. One patient was under follow-up and treatment because of hypertension (HT) that started before pregnancy and three patients were under follow-up due to hypertension that started during pregnancy. There was no known additional disease in one patient. The delivery week of the patients was between 28 and 34 weeks of gestation. Pathological laboratory values were most frequently seen in LDH, albumin, and protein values. Every patient was discharged with outpatient follow-up. Epilepsy continues in one patient, HT in two patients, and isolated nephropathy in one patient after PRES.
Conclusion: PRES should be considered especially in pregnant women with neurological symptoms including visual impairment and headache. Clinical suspicion and neuroimaging are required for the diagnosis of PRES.
Key words: PRES, eclampsia, blindness, visual changes

ÖZ

Amaç: Bu çalışmanın amacı posterior reversible ensefalopati sendromunun (PRES) risk faktörlerini ve klinik gidişatı değerlendirmektir.
Gereç ve Yöntem: Bu çalışmada 2017-2022 yılları arasında tersiyer bir sağlık merkezinin acil kadın doğum polikliniği ve yoğun bakım ünitesinde PRES tanısı almış gebe veya lohusalara ait veriler retrospektif olarak incelendi. Tüm hastalar kadın doğum, nöroloji, göz, radyoloji ve yoğun bakım hekimleri tarafından değerlendirildi, kan tahlilleri ve görüntülemeleri aynı sağlık kuruluşunda yapıldı. Her hastanın başvuru şikâyetleri, laboratuvar değerleri, görüntüleme yöntemleri, eşlik eden hastalıklar, doğum şekli ve doğum sonrası dönemi ile ilgili verileri kaydedildi.
Bulgular: Beş yılda görüntüleme yöntemleri ve klinik bulgulara göre toplam yedi hastaya PRES tanısı kondu. Bu hastalardan birinde üç yıl arayla iki kez PRES gelişmişti. Altı hasta eklampsi ile başvurdu. Bir hastaya postpartum ilk haftada, diğer hastalara gebelikte tanı kondu. Hastalardan 42'sinde bulanık görme, 2'sinde körlük varken 1 hastada görme şikâyeti yoktu. Hastaların 3 tanesinde yeni başlayan mood değişiklikleri (bir hastada konfüzyon, iki hastada ajite) vardı. Ek hastalık sorgulamasında hastalardan birinde bilinen diabetes mellitus (DM) vardı oral antidiyabetik kullanıyordu. Bir hasta gebelik öncesi başlayan hipertansiyon (HT), üç hasta gebelikte başlayan hipertansiyon nedeniyle takip ve tedavi altındaydı. Bir hastada bilinen ek hastalık yoktu. Hastaların doğum haftası 28-34 gebelik haftaları arasındaydı. En sık LDH, albümin ve protein değerlerinde anormal kan düzeyi saptanmıştı. Tüm hastalar ayaktan taburcu edildi. PRES sonrası bir hastada epilepsi, iki hastada HT ve bir hastada izole nefropati varlığı devam etmişti.
Sonuçlar: PRES özellikle görme bozukluğu ve baş ağrısı gibi nörolojik semptomları olan gebe ve lohusalarda akla gelmelidir ve tanı için nörogörüntüleme gereklidir.
Anahtar Kelimeler: PRES, eklampsi, körlük, görme bozukluğu

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Submitted/Başvuru: 29.12.2022 • **Revision Requested/Revizyon Talebi:** 02.01.2023 • **Last Revision Received/Son Revizyon:** 07.01.2023 • **Accepted/Kabul:** 20.02.2023 • **Published Online/Online Yayın:** 25.04.2023

INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) is a clinical radiological diagnosis that is accompanied by neurological complaints most commonly affecting the posterior cerebral area, and symmetrical vasogenic edema in the parieto-occipital region, classically seen on cerebral CT and MRI imaging. This was first described in 1996. PRES is also known as acute hypertensive encephalopathy or reversible posterior leukoencephalopathy. Acute HT causes different cortical changes in MRI than in chronic HT patients. As the cases reported in the literature increased, it was observed that the posterior frontal and temporal lobes and even the cerebellum were involved. In the syndrome, which is initially known as reversible, clinical complaints are usually temporary, but there is also literature showing an increased risk of stroke in these patients in the long term (1,2).

The most common neurological complaints include visual disturbances, headaches, convulsions, and confusion.

The pathogenesis of PRES is not clear, but hypertension, preeclampsia/eclampsia, renal failure, autoimmune diseases, and the use of cytotoxic agents are risky for PRES. It is more common in women than men (3). Preeclampsia or eclampsia are present in 7-20% of PRES cases (4). According to one opinion, PRES is a part of the pathogenesis of eclampsia (5). In the acute phase of the disease, neurological complaints (vision loss (sometimes even Anton's syndrome), visual aura, hemianopia, visual hallucinations, headache, cognitive changes such as drowsiness, confusion, agitation, coma, convulsions, nausea, regional muscle weakness, sensory disturbances, speech deterioration) are seen (6). In the fundoscopic examination of the eclamptic patient, papilledema, flame-shaped hemorrhage, and exudation can be seen (7).

In the treatment of PRES, the risk of complications and seizures can be reduced with agent-directed treatment and supportive ICU treatments.

With this study, we wanted to discuss the presentation, risk factors, and clinical course in PRES cases.

MATERIAL and METHODS

The data of the patients were obtained from the tertiary health center Gynecology and Obstetrics Clinic and intensive care unit records with the approval of the ethics committee dated 10/11/2022, numbered 953.

Age, gravida, comorbidities (DM, thyroid diseases, HT, kidney diseases), symptoms, relapse status, mood changes, obstetric history, complications, arterial blood pressure at admission, neurology/ophthalmology/radiology, and reanimation unit study records of the patients were examined and information was recorded.

The diagnosis of PRES was made with the joint evaluations of obstetrics, radiology, and neurology.

MRI examinations were performed on either a Siemens Amira 1.5 Tesla or Philips Ingenia 1.5 Tesla (All MRI studies included T2-weighted, T2 FLAIR, DWI, and SWI).

Albumin, total protein, liver function tests (Ast, Alt, Ldh), RDW, and blood sugar results were compared in the blood when the application was made with clinical complaints from the laboratory values. In cases of repeated PRES, the values in both cases were recorded separately.

RESULTS

There were seven patients in total, one of whom was a patient who had PRES for the second time, three years apart.

While one patient had PRES in the previous pregnancy, there was no PRES in the next pregnancy.

The patients were between the ages of 21 and 33. Two patients were nulliparous, and four were multiparous.

Of the six patients, one was from Turkmenistan, one was Moroccan, one was Syrian, and three were Turkish.

One patient had PRES postpartum during week 1 and the others had PRES during pregnancy.

Only one patient had known HT before pregnancy and was using regular medication. HT was detected for the first time in three patients when they were pregnant or presented with seizures.

In the case of recurrent PRES, the first PRES developed after delivery, and the second PRES developed before delivery. She did not have any known systemic disease in her pregnancy, which was her first PRES. In the second attack, she had HT that continued after the first PRES. In the recurrent PRES case, blurred vision developed in the first attack, and blindness developed in the second attack.

All patients complained of headaches.

One patient had PRES in her first pregnancy, and her pregnancy was uneventful one year later.

Of the seven patients, two had blindness and three had blurred vision. One patient had only convulsions without vision problems.

The patients delivered at between 28 and 34 weeks of gestation. All babies were discharged without any problem after the neonatal intensive care process.

All seven patients had generalized tonic-clonic seizures, but there was no seizure in the second attack of the recurrent PRES case.

Agitation developed in two patients and confusion in one patient. There was no mood change in the other patients.

One patient had gestational DM. Her blood sugar was normal at the time of admission, and it never increased afterward (Table 1).

Table 1: Demographic-clinical characteristics of the patients

	Age-Gestational week	Comorbidity	COVID	Gravida	TA (mmHg)	Onset time	Headache	Eclampsia	Visual disturbance	Mood changes	Complication	Recurrence
Case 1	27 y, 33 gw	GHT	None	G3	180/110	At birth	+	+	Blurred vision	None	Ongoing HT	First
Case 2	30 y, 33 gw	HT	None	G4	130/80	At birth	+	–	Blindness	None	Ongoing proteinuria	Second
Case 3	31 y, 30 gw	GHT, obesity	None	G4	140/70	At birth	+	+	Blurred vision	Confusion	For 6 months HT	None
Case 4	25 y, 32 gw	GHT	None	G3	160/100	At postpartum 1st week	+	+	Blurred vision	None	For 6 months HT	None
Case 5	30 y, 30 gw	None	None	G3	150/70	At birth	–	+	None	Agitation	Epilepsy	None
Case 6	21 y, 28 gw	GHT	None	G1	180/110	At birth	+	+	Blurred vision	None	1 month proteinuria	None
Case 7	33 y, 34 gw	GDM	None	G1	137/80	At birth	+	+	Blindness	Agitation	None	None

GHT: Gestational hypertension, GDM: Gestational Diabetes Mellitus, HT: Hypertension

The patient whose albumin and protein levels were extremely low at admission, and who received DIC treatment in the ICU, had cerebral infarction areas and epilepsy that started after a PRES attack and has been continuing for five years.

All patients were delivered with SCA due to either eclampsia or severe preeclampsia, and all clinical complaints regressed within one to two days after delivery.

In biochemical tests, every patient had elevated LDH and low albumin and total protein. There were elevated liver function tests in some patients. RDW was normal in all the patients.

The nephrology follow-up of one patient continues due to proteinuria.

HT, which required six months of medication, continued in two of the patients (Table 2).

DISCUSSION

With our study results, we determined that PRES is a recurring syndrome with permanent neurological complications in the long term, and we think that the diagnosis of PRES can be made more frequently if cranial imaging is performed in every pregnant woman with neurological findings.

Despite the large population and high number of births in the tertiary health center where the study was conducted, the fact that only seven patients (one case being recurrent) were diagnosed with PRES in five years may be due to the fact that the diagnosis could not be made. According to the study by Chao et al. in the literature, the majority of patients with eclampsia actually have PRES, but when the patient has convulsions, the diagnosis can be made less frequently than necessary because the diagnosis is made with neurological imaging and evaluations. Especially in women with severe preeclampsia and headache, if MRI is performed, it can be detected at a higher rate (8).

PRES may start secondarily to many pathologies such as hyperperfusion and arterial extravasation, vasogenic edema and hypoperfusion, endothelial dysfunction, fluid overload, and metabolic changes. The main problem in preeclampsia and eclampsia is thought to be related to endothelial damage. Supporting this theory is the increase in the secretion of substances such as fibrinonectin, TPA, thrombomodulin, endothelin1, and VWF that can damage the endothelium in preeclampsia, and high LDH and erythrocyte morphology changes indicating endothelial damage (9). In our study, LDH values were high in all the patients, and RDW was within physiological values in all the patients.

Albumin and total protein levels were low in all patients. There are results in the literature showing that low albumin levels in adults may be a risk factor for PRES (10). AST, ALT was elevated in only two patients. Blood glucose levels were variable as viewed from the spot. Hemoglobin, urea, and creatinine levels were also normal in the patients. However, we think that the laboratory values of the patients did not deteriorate much because they were delivered close to the onset of symptoms.

In healthy people, there is a mechanism that protects the brain from systemic blood pressure changes. However, in cases such as sudden increased blood pressure or excessive fluctuation, autoregulation may be impaired, and the blood-brain barrier becomes disrupted with an increase in systemic arterial blood pressure, with blood flow in the brain increasing and arterial extravasation occurring in the cerebral parenchyma (11,12).

Table 2: Laboratory results of the patients

	Albumin-total protein (3.5-5.2)-(6.6-8.3) gr/dl	LDH (1-248) U/L	AST-ALT (1-35) U/L	RDW (11-16) %	Blood sugar (74-106) mm/dl
Case 1	2.35-5.05	1771	113-33	14.3	119
Case 2	3.27-6.22	264	20-12	13.3	129
Case 3	3.18-5.47	419	25-11	12.5	144
Case 4	3.3-5.98	291	21-14	15.6	80
Case 5	0.6-3.1	574	24-11	13.3	119
Case 6	2.18-5.56	383	26-12	12.5	92
Case 7	2.46-4.67	357	143-122	14.1	88

LDH: Lactate dehydrogenase, AST: Aspartate aminotransferase, ALT: Alanin aminotransferase, RDW: Red blood cell distribution width

Although increased perfusion is seen more frequently on MRI in PRES cases, hypoperfusion may be seen in some patients because in some cases, disruption of autoregulation causes hypoperfusion and cytotoxic edema and cerebral local infarcts. Cerebral infarcts may also occur due to vasogenic edema and compression of the edema. This allows vasoconstriction findings to be seen on MRI. In one of our patients, who developed permanent epilepsy complications, edematous areas due to PRES as well as cerebral infarct areas were observed.

PRES is most common in posterior cerebral arterial circulation areas (parieto-occipital region) with weaker sympathetic innervation, which are less tolerant of edema and vascular changes that occur following the destruction of the blood-brain barrier. Therefore, vasogenic edema and related sensory changes, seizures, and vision loss are the most common complaints (13).

MRI is superior to CT in imaging (14). However, in an MRI performed very close to the onset of symptoms, the diagnosis may not be made because the signs of vasogenic edema are not fully established yet. In addition, the possibility of the disappearance of symptoms and radiological findings until the patient is delivered and stabilized due to concomitant obstetric emergencies (eclampsia, severe preeclampsia, fetal distress, etc.) may also cause the diagnosis to be missed. PRES symptoms usually disappear very quickly, even with a reduction in blood pressure and mild supportive care, and the patient may not have had a control radiological evaluation after the initial MRI, which is normal. Another problem that should not be forgotten is the inadequacy of the radiological method or team. MRI findings may not be as typical, or the sensitivity of the device used may be weak. ASL (arterial spin labeling)-based perfusion is a new marker for the diagnosis of PRES, even in the absence of vasogenic edema (15). We diagnosed all seven patients with MRI, but since we did not have ASL in our hospital, we were able to use a 1.5 Tesla MRI.

Cerebral MRI findings in PRES are symmetrical and reversible in almost every patient. All of our patients had involvement of the parieto-occipital region. There was also involvement in the frontal area in two patients and at the basal ganglia and pons level in one patient. We believe that a small number of patients were diagnosed because MRI findings may not be established in the early period and the findings quickly disappear in the late period.

Although it is known that the COVID-19 infection, which started in 2019 and led to a pandemic, may increase the frequency of PRES through a cytokine storm, vascular permeability changes, dysregulation in the renin-angiotensin system, and the drugs used (hydroxychloroquine and tocilizumab), we found that the patients at least did not have a diagnosed COVID-19 infection (16).

Visual disturbances are seen in 26-67% of patients and cortical blindness in 8-33% in PRES (17). They described blindness in two of the patients and blurred or impaired vision in four. There was no visual impairment in the patient who later developed neurological complications.

PRES is typically associated with severe hypertension, but it may also occur with rapid increases in blood pressure in patients with endothelial damage and in patients with only mildly elevated blood pressure (18). PRES may also develop in normotensive or hypotensive patients (19). TA was high in all of our patients.

The literature warns that having PRES may cause ischemic brain damage, permanent neurological deficit, relapse, or death (20,21). Permanent brain damage, recurrence, or death may not be present in PRES that are intervened in a timely and correct manner (22). In cases where adequate and rapid treatment is not performed, epilepsy becomes permanent most frequently (23). In fact, permanent neurological damage is related to ischemia and/or intracranial hemorrhage. In such cases, the mortality rate is 15% (24,25). AntiPL and thrombophilia scans are clean, but MRI results suggestive of cerebral infarction are detected in our PRES cases, which have convulsions despite still using antiepileptic drugs.

PRES is a rare clinical syndrome that can occur for reasons other than pregnancy. Clinical complaints usually resolve rapidly after PRES and there are no sequelae. One of the patients in the study has epileptic seizures that started and continued after PRES. Two patients had HT for six months, and one patient had persistent proteinuria. Headache and visual disturbances in

every patient resolved completely in one to two days.

In the literature, the rate of PRES being reversible is between 35% and 100%. In reversible cases, sometimes there is a complete recovery, and sometimes there is a partial recovery (26). Among patients with a follow-up CT or MRI, 49% to 75% have a resolution of the initial abnormalities within five days to seventeen months (27).

The most critical treatment is the regulation of blood pressure. However, an aggressive TA reduction is not recommended because it decreases cerebral perfusion. It is recommended to keep it at systolic 130-150/diastolic 80-100 mmHg. It would be correct to avoid nimodipine, diazoxide, and ketanserine in the treatment of HT. It is suspected that nifedipine may increase angioedema. Nicardipine and labetalol are suitable. No additional treatment is recommended for convulsions only if status epilepticus is present (28).

The most important limitation of the study is the small number of patients and the absence of late-term and control MRI.

According to the results of our study, PRES should be considered in pregnant women with neurological findings including headache and visual impairment, especially if they have preeclampsia/eclampsia. Because PRES is a clinical entity that can recur and cause permanent neurological deficits, MRI should be used more liberally to diagnose it and start treatment as early as possible.

Ethics Committee Approval: This study was approved by Istanbul Medipol University Non-Interventional Clinical Research Ethics Committee (Date: 10.11.2022, No: 953).

Peer Review: Externally peer-reviewed.

Author Contributions: Conception/Design of Study- Ö.K.A., E.O., H.G.; Data Acquisition- Ö.K.A., E.E.K., G.E.A., M.S.S.; Data Analysis/Interpretation- Ö.K.A., N.K.A., N.H.; Drafting Manuscript- Ö.K.A., N.K.A., E.E.K., M.S.S.; Critical Revision of Manuscript- H.G., Ö.K.A., E.O., G.E.A., N.H.; Final Approval and Accountability- Ö.K.A., E.O., H.G., E.E.K., G.E.A., M.S.S., N.K.O., N.H.; Material and Technical Support- Ö.K.A., M.S.S.; Supervision- H.G., N.K.A., N.H.

Conflict of Interest: The authors have no conflict of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

REFERENCES

1. Parauda SC, Zhang C, Salehi Omran S, Schweitzer AD, Murthy SB, Merkler AE,et al. Risk of stroke after posterior reversible encephalopathy syndrome. Stroke 2022;53(11):3313-9.
2. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A.,et al. A reversible posterior leukoencephalopathy syndrome. N Engl J Med 1996;22;334(8):494-500.
3. Bartynski WS. Posterior reversible encephalopathy syndrome, part 1: fundamental imaging and clinical features. AJNR Am J Neuroradiol 2008;29(6):1036-42.
4. Tek ŞÇ, Uyar AŞ, Çakıcı Z, İnal MT, Memiş D, Tekataç A, et al. Posterior reversible ensefalopati sendromu: iki olgunun sunumu. Turk J Intensive Care 2019;17:44-8.
5. Martin JN, Jr Brewer JM, Blake PG, Owens MY, LaMarca B. PP137. Posterior reversible encephalopathy syndrome (PRES) is a constant component of eclampsia. Pregnancy Hypertens 2012;2(3):314.
6. Triplett JD, Kutlubaev MA, Kermode AG, Hardy T. Posterior reversible encephalopathy syndrome (PRES): diagnosis and management. Pract Neurol 2022;22(3):183-9.
7. Stott VL, Hurrell MA, Anderson TJ. Reversible posterior leukoencephalopathy syndrome: a misnomer reviewed. Intern Med J 2005;35(2):83-90.
8. Chao AS, Chen YL, Chang YL, Chao A, Su, SY, Wang TH. Severe pre-eclamptic women with headache: is posterior reversible encephalopathy syndrome an associated concurrent finding? BMC Pregnancy Childbirth 2020;1;20(1):336.
9. Schwartz RB, Feske SK, Polak JF, DeGirolami U, Iaia A, Beckner KM, et al. Preeclampsia-eclampsia: clinical and neuroradiographic correlates and insights into the pathogenesis of hypertensive encephalopathy. Radiology 2000;217(2):371-6.
10. Furtado A, Hsu A, La Colla L, Zuccoli G. Arterial blood pressure but not serum albumin concentration correlates with ADC ratio values in pediatric posterior reversible encephalopathy syndrome. Neuroradiology 2015;57(7):721-8.
11. Strandgaard S, Paulson OB. Cerebral autoregulation. Stroke 1984;15(3):413-6.
12. Rabinstein AA, Mandrekar J, Merrell R, Kozak OS, Durosaro O, Fugate JE. Blood pressure fluctuations in posterior reversible encephalopathy syndrome. J Stroke Cerebrovasc Dis 2012;21(4):254-8.
13. Akgün N, Karaman M, Başyiğit S, Yılmaz H, Özcan AA. Posterior reversible ensefalopati: 2 olgu sunumu. İstanbul Tıp Dergisi 2010;11(2): 82-5.
14. Doelken M, Lanz S, Rennert J, Alibek S, Richter G, Doerfler A. Differentiation of cytotoxic and vasogenic edema in a patient with reversible posterior leukoencephalopathy syndrome using diffusion-weighted MRI. Diagn Interv Radiol 2007;13(3):125-8.
15. Fazeli S, Noorbakhsh A, Imbesi SG, Bolar DS. Cerebral perfusion in posterior reversible encephalopathy syndrome measured with arterial spin labeling MRI. Neuroimage Clin 2022;35:103017.
16. Donaldson L, Margolin E. Posterior Reversible Encephalopathy Syndrome in Acute COVID19 Pneumonia. Can J Neurol Sci 2022;49(6):791-2.
17. Vandenbossche G, Maquet J, Vroonen P, Lambert G, Nisolle M, Kridelka F, et al. A reversible posterior leucoencephalopathy syndrome including blindness caused by preeclampsia. Facts Views Vis Obgyn 2016;8(3):173-7.
18. Eastabrook G, Brown M, Sargent I. The origins and end-organ consequence of pre-eclampsia. Best Pract Res Clin Obstet Gynaecol 2011;25(4):435-47.
19. Granata G, Greco A, Iannella G, Granata M, Manno A, Savastano E, Magliulo G. Posterior reversible encephalopathy syndrome--Insight into pathogenesis, clinical variants and treatment approaches. Autoimmun Rev 2015;14(9):830-6.
20. Ferraldeschi M, Tari Capone F, Di Lisi F, Patella R, Ceschim V, Cao M, et al. When a pregnancy required a neurological consultation: a case report. Clin Ter 2012;163(6):487-90.
21. Vandenbossche G, Maquet J, Vroonen P, Lambert G, Nisolle M, Kridelka F, et al. A reversible posterior leucoencephalopathy syndrome including blindness caused by preeclampsia. Facts Views

Vis Obgyn 2016;8(3):173-7.

22. Pizon AF, Wolfson AB. Postpartum focal neurologic deficits: posterior leukoencephalopathy syndrome. J Emerg Med 2005;29(2):163-6.
23. Burnett MM, Hess CP, Roberts JP, Bass NM, Douglas VC, Josephson SA. Presentation of reversible posterior leukoencephalopathy syndrome in patients on calcineurin inhibitors. Clin Neurol Neurosurg 2010;112(10):886-91.
24. Servillo G, Bifulco F, De Robertis E, Piazza O, Striano P, Tortora F, et al. Posterior reversible encephalopathy syndrome in intensive care medicine. Intensive Care Med 2007;33(2):230-6.
25. Lee VH, Wijdicks EF, Manno EM, Rabinstein AA. Clinical spectrum of reversible posterior leukoencephalopathy syndrome. Arch Neurol 2008;65(2):205-10.
26. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A, et al. A reversible posterior leukoencephalopathy syndrome. N Engl J Med 1996;334(8):494-500.
27. Casey SO, Sampaio RC, Michel E, Truwit C.L. Posterior reversible encephalopathy syndrome: utility of fluid-attenuated inversion recovery MR imaging in the detection of cortical and subcortical lesions. AJNR Am J Neuroradiol 2000;21(7):1199-206.
28. Vandenbossche G, Maquet J, Vroonen P, Lambert G, Nisolle M, Kridelka F, et al. A reversible posterior leucoencephalopathy syndrome including blindness caused by preeclampsia. Facts Views Vis Obgyn 2016;8(3):173-7.