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Review Article

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Delayed Post-partum Eclampsia or PRES: An Enigmatic Connection in Maternal Health

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Abstract

Posterior reversible encephalopathy syndrome (PRES) is a neurological condition that is identified by headache, seizures, vomiting and nausea as well as distorted vision and in many cases blindness. It mainly affects women specifically females with eclampsia. Our study throws light on assessing cases of PRES diagnosed in postpartum eclampsia with 48 hours after delivery occurrence being described as late postpartum. What specific pathophysiological processes and risk factors lead to the onset of posterior reversible encephalopathy syndrome (PRES) in cases of postpartum eclampsia, and what role does early diagnosis and intervention play in increasing maternal survival? A literature review was conducted after comprehensive search from PubMed and Google Scholar from 2013 to 2024. Finally, 12 studies were taken for analyzing. Occurrence of PRES in the age group 20-35 with headache was most common (33%) and preeclampsia history (83.3%) was another key feature. Seizure was identified in 75% and PRES was diagnosed in some cases without seizures through MRI. PRES was developed in six post-partum cases post caesarean. Treatment consisted of $MgSO_4$ alone given to 50% cases and $MgSO_4$ with phenytoin given to 25% of cases. Even though medical professionals might be familiar with risk factors of eclampsia and its symptoms, they should also be aware of PRES development in patients with eclampsia. Headache has been identified as one the key drivers and crucial symptom. An early implementation of MRI is vital for timely diagnosis of PRES.

Keywords: ***Posterior reversible encephalopathy syndrome, post-partum eclampsia, Headache, MRI, Clinical features, Maternal health.***

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a unique neurological disorder characterized by headache, vomiting, nausea, blindness and seizures. The incidence of PRES is found in females mostly in eclamptic patients. The late post-partum is those that occur 48 hours after delivery. Its incidence is 5-26% of all eclampsia patients. Eclamptic fits occur during pregnancy if it is antepartum and if it occurs in labor it is intrapartum. If the the fits occur after delivery it is termed as post partum. The incidence of eclampsia is 1 to 5% in India and in all developing countries [1]. PRES as the name suggests is a totally reversible condition wherein all the neurological disorders are not found [2]. The patient may however may have a history of new onset of hypertension. We are analyzing the patients in whom PRES was diagnosed as postpartum eclampsia [3]. PRES is not only associated with obstetric cases of eclampsia but also noted among patients with history of having auto immune diseases like systemic lupus erythematosus, sclera, derma patients with transplantation of organs, renal disease, hyper calcamia, thrombocytopenia, henoch schonlein purpura - immunosuppressive drugs like ciclosporin. The pathogenesis of PRES is an enigma and still a debate [4]. There are theories explaining it. There is vasoconstriction following severe hypertension and the extravasation of fluid into the brain tissue at the periphery due to the vasogenic edema caused by toxins causing endothelial cells damage [5]. Another theory says there is cerebral vasoconstriction leading to decrease in blood flow thereby resulting in thrombosis leading to cerebral ischemia. It has been suggested that vasoconstriction leads to hypoperfusion and edema. This is done by T lymphocytes. When the treatment is done in time, most of the patients have complete recovery from neurological disorder and within two weeks there is resolution of the radiological lesions which were shown in MRI depicting white matter hypodensities in parieto-occipital regions [6]. CT is an investigation of choice in PRES but MRI is a gold standard in diagnosis and FLAIR (Fluid Attenuated Inversion Application Recovery) [7]. This shows a typical hyper intensity in white matter of the occipital lobe, parietal lobe with peripheral edema. In this study, we insist that in all patients diagnosed as eclampsia who develop neurological disorder, it is mandatory to rule out PRES. The early diagnosis and treatment is the cornerstone for complete recovery. This review will discuss the clinical features of PRES in postpartum eclampsia and emphasizes the importance of early diagnosis of this condition at the time of eclampsia for the prevention of neurological morbidity.

Materials and methods

The search was done in PubMed and Google Scholar to find out PRES related to post-partum eclampsia from the time period 2013 to 2024.

The present systematic review and meta-analyses were done as per Preferred Reporting Item for Systematic Review and Meta Analyses guidelines (**Figure 1**) [8]. Two researchers namely A.T.C

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and S.S. conducted a literature search in PubMed and Google Scholar for the period from 2013 to 2024 using the keywords "Posterior Reversible Encephalopathy Syndrome", "Post-partum eclampsia" and "PRES". The search included peer-reviewed journals and related retrieved publications. The inclusion criteria included cases available with complete data, post-partum eclampsia cases associated with PRES and articles published in English. The excluded studies included studies with abstracts only available wherein full PDFs were not available, case series and reports of antepartum pre-eclampsia and eclampsia cases with PRES, the non-obstetric cases and other risk factors for PRES like systemic lupus erethamatosus, drugs, cancer, immunosuppressant, sepsis, hypertension cases.

A comprehensive search was done on basis of criteria and full text was analyzed. The quality of the articles was analyzed by using New Castle Ottawa assessment scale [9]. Finally, a total of 12 studies matched the quality of assessment and were selected for the further research. The first author, type of study, year of publication, sample size and demographic analysis, clinical features, comorbidities and the diagnosis of PRES were tabulated. Microsoft Excel 2016 was used to tabulate data and R Studio for preparation of graphs.

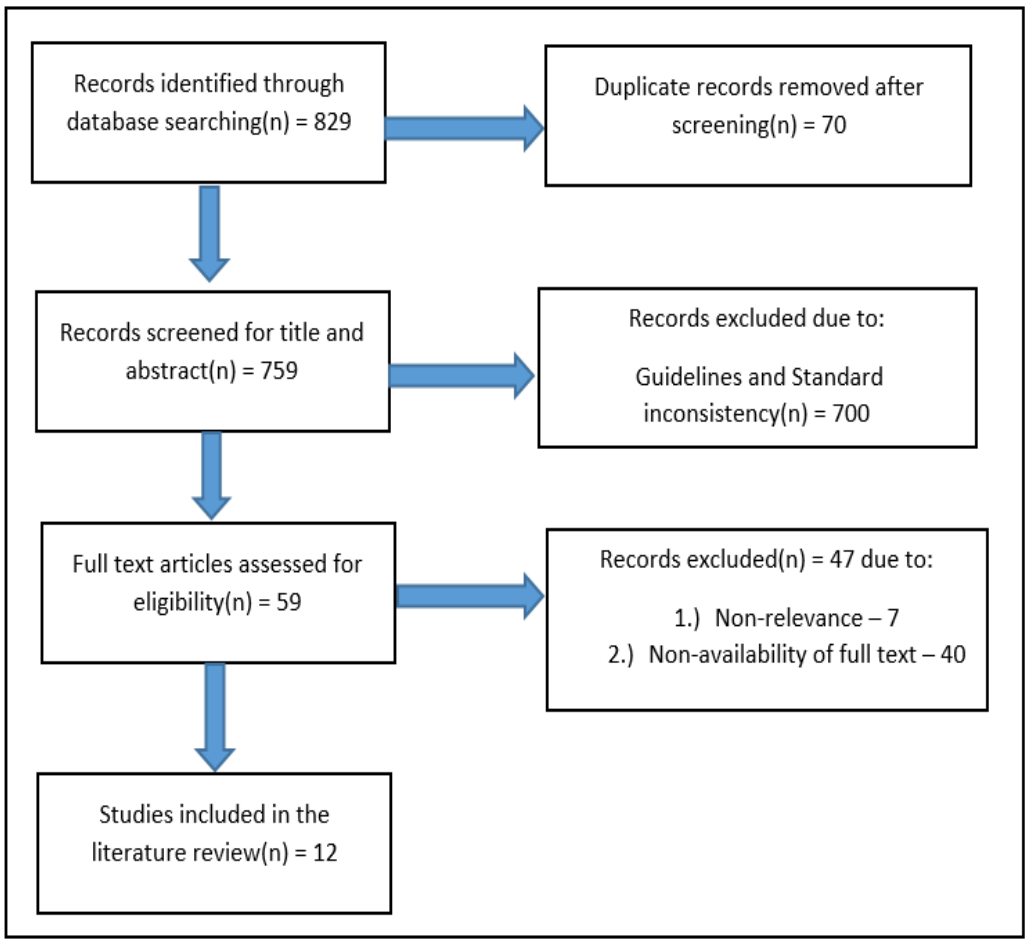

Figure 1

Results

According to the search a total of 829 articles were retained in target database. Seventy duplicate articles were removed. The remaining 759 articles were screened for title and abstract out of which 700 articles didn't meet eligibility and were removed due to guidelines and standard inconsistency. Finally, 12 articles were determined to be included in the analyses. The demographics and other clinical features, diagnostic methods and investigations in various studies were tabulated (**Table 1 and 2**).

The age group 20-35 showed an overall of prevalence rate 33% but 17% under 25 years (**Table 3 and Figure 2**). Multiparous and prim parous patients had equal prevalence with the youngest prim parous in our study was 16 years old (**Table 4 and Figure 3**). Among the clinical features, headache was noted in the majority showing a 92%. Visual disturbances and blindness both showed 42%. Vomiting and nausea was noted in 33%. Proteinuria was seen in 33%. History of preeclampsia was noted in 10 patients in our study indicating a prevalence of 83.3% (**Table 5 and Figure 4**). HELLP syndrome was noted in one patient with an 8% (**Table 6 and Figure 5**). The patients complaints and treatment given in each study author-wise were tabulated (**Table 7**). Two patients had GDM showing 16.67% and one had DM contributing to 8.3%. One patient had right sided hemiparesis showing prevalence of 8.3%. Out of 12 patients, 3 patients did not exhibit seizures making a prevalence of 25% but MRI showed typical PRES since they had loss of vision and severe headache and high BP. One patient had no h/o BP but showed diminution of vision in both eyes, severe headache, vomiting and nausea and seizures and was diagnosed by MRI. The same patient had a h/o taking NSAIDs and exhibited lupus anticoagulant factor and had bronchial asthma17. All the 9 patients who had seizures showed brisk tendon reflexes and Kernig's sign was negative. Out of 12 post-partum patients, six had PRES following LSCS. In our study we found PRES developing in the post-op caesarean patients on day one, two, four, five, seven and eight. The treatment protocol consisted of 50% of $MgSO_4$ alone and 25% of combination of $MgSO_4$ + phenytoin (**Table 8 and Figure 6**).

Table 1: Demographics and clinical characteristics of patients with PRES

Sl No	Author	Year	Country	Age	BP	Day of Onset	Day of Discharge
1	Maïga Youssoufa et al[10]	2013	Mali	39	240/120	12 hrs after NVD, recurrence 9 months later	Not mentioned
2	Ülkü Mete Ural et al[11]	2014	Turkey	33	210/120	13 hrs after delivery	6th postpartum day
3	Pooja Gupta Jain et al[12]	2015	India	20	200/130	4 days after delivery	10th day
4	Makarim et al[13]	2016	Sri Lanka	33	200/110	8th day after LSCS	11th day
5	Maasoumeh Mirazamoradi et al[14]	2017	Iran	16	160/110	2 hours after surgery	7th day
6	Antonio Sesar et al[15]	2018	Mostar, Bosnia and Herzegovina.	35	195/110	1st postoperative day	After 3 days
7	Kaori Masai et al[16]	2019	Japan	23	141/103	73 days from delivery	Not mentioned
8	Majumder A et al[17]	2020	Bangladesh	19	130/80	7 days after LSCS	6th day
9	Niruby Rasendrakumar[18]	2021	India	29	140/90	5 days after delivery	Not mentioned
10	Suman Nishad et al[19]	2022	India	23	180/110	6 days after still born delivery	"
11	Manmin Zhu et al[20]	2023	China	36	166/65	5th day from LSCS	"
12	Anh Dinh Bao Vuong et al[21]	2024	Vietnam	31	200/120	2nd day from LSCS	-"

NVD: normal vaginal delivery; LSCS: lower segment caesarean section

Table 2: Author-wise investigations and diagnoses of PRES cases

Sl No	Author	Investigations	CT/MRI
1	Maïga Youssoufa et al [10]	Proteinuria and other biological tests like complete blood count, prothrombin time, liver enzymes, blood urea and creatinine, blood glucose level, serum electrolytes-Normal	CT Bilateral symmetric occipital hematoma of parieto-occipital region with peripheral edema
2	Ülkü Mete Ural et al [11]	CBC- HgB - 9.1g/Dl, Platelet count- 162.000/mm^3, KFT, LFT, ECG-normal	MRI -Hyperintense and FLAIR signal lesions extending beyond the occipital lobes and involved both cerebellar hemispheres
3	Pooja Gupta Jain et al [12]	HgB 7.8 gm%, platelet 2.89 lakh per cumm, urine albumin 3 plus and rest of the investigations (liver function test, renal function test, coagulation profile) within normal range	MRI- hyper intensity in left posterior parietal and occipital lobe, involving left basal ganglia and right caudate nucleus in T2 weighted image
4	Makarim et al [13]	D-dimer (200ng/dl), Serum Calcium (7.9mg/dl), ANA (Negative), Serum Magnesium (2.2mg.dl), cardiolipin antibodies (Negative) and clotting profile-normal. urine albumin: 1+	MR Arteriogram & MR Venogram) -bilateral symmetrical cerebral ischemia/ vasogenic edema of cortical & sub-cortical regions of parieto-occipital areas and water shed areas of frontal lobes. normal funduscopy with normal CT scan of Brain
5	Maasoumeh Mirazamoradi et al [14]	Hgb: 13.1%, Platelet count: 222000 per microliter, Urine Protein: 4+. Liver function test, kidney function test, electrolytes and clotting parameters were normal	MRI: abnormal signal intensity in the superior area of both occipital lobes and similar changes in the right frontal lobe
6	Antonio Sesar et al [15]	The complete blood count, liver function tests, clotting parameters, and electrocardiogram: normal. Urine analysis revealed proteinuria 2+.	MRI T2 weighted FLAIR showed hyper intensive signals in parietal and occipital regions
7	Kaori Masai et al [16]	Lumbar puncture showed 1lymphocyte/mL and cerebrospinal f fluid protein of 86mg/dL. Beta-2-microglobulin, angiotensin converting enzyme (ACE), and Mycobacterium tuberculosis DNA were negative. Blood examination presented leukocytosis (11,220/mL), low folic acid (2.10ng/mL), and hypopotassemia (2.9mmol/L). No serological evidence of acute infection with the viruses. ACE and b-D glucan were negative. Autoimmune antibodies listed were all negative.	MRI T2 weighted FLAIR: bilateral pareito-occipital and frontal lobe, hyper intense signals, MR angiography - no spasm
8	Majumder A et al [17]	white cell count of 19000, N-85.4%, haemoglobin 10.7, platelets 334000. CRP 55.26.Urinalysis: 3+protein and plenty of pus cells and RBC 12-15/HPF. Phase contrast microscopy: 2% of dysmorphic RBC in urine. No Casts were present. PT, PTT, INR and liver functions, renal functions including serum electrolytes were within normal limits. ANA was found positive with 25 u/ml while anti dS DNA was negative. Lupusanti coagulant: +ve	CT brain: infarction in both occipital and posterior parietal lobe characteristic of posterior reversible leukoencephalopat-hy syndrome

9	Niruby Rasendrakumar [18]	lactate dehydrogenase level of 243 U/L, uric acid 3.9 mg/dL, 24-hour fluid intake of 2950 mL and 24-hour urine output of 2850 mL. Urinalysis revealed a urine protein to creatinine ratio of 3.81 g/gCr. Capillary blood glucose was low at 25 mg/dL. Red blood cell count, platelet count, hemoglobin levels, renal function, and coagulation panel were all within the normal range. Fundoscopy revealed no evidence of papilledema.	MRI T2 weighted FLAIR showed hyperintensive signals in parietal, occipital and frontal regions, angiogram revealed no abnormalities
10	Suman Nishad et al [19]	Basic investigations might have been done. USG pelvis showed placental tissue	T2 imaging showed bilaterally posterior parieto-occipital hyper densities in the area of cortex and subcortical white matter.
11	Manmin Zhu et al [20]	routine hematuria myocardial enzyme, liver and kidney function, coagulation function, antinuclear antibody, rheumatoid factor virus, no obvious abnormalities found.	MRI T2 weighted FLAIR showed hyper intensive signals in parietal, occipital and frontal regions
12	Anh Dinh Bao Vuong et al [21]	renal function test was normal, except for a serum acid uric of 509 (µmol/l). The lactate dehydrogenase (LDH) concentration was measured at 2514 U/L. Glycemia was normal. The coagulation profile showed a weakly contractile blood clot. Liver function test impaired and thrombocytopenia present. Pleural effusion was present due to hypoproteinemia. HELLP syndrome noted.	MRI T2 weighted FLAIR: Bilateral parieto-occipital lobe and lenticular nucleus suggesting PRES. MR angiography- no malformation

CT: computed tomography; CBC: complete blood count; HgB: haemoglobin; KFT: kidney function tesr; LFT: liver function test; ECG: electrocardiogram; FLAIR: fluid attenuated inversion recovery; MRI: magnetic resonance imaging; USG: ultrasonography; HELLP: hemolysis, elevated liver enzymes low platelet

Table 3: Age-wise distribution of PRES patients

Sl No	Age group	Total number	Percentage
1	<20	2	17
2	20-30	4	33
3	31-35	4	33
4	>35	2	17

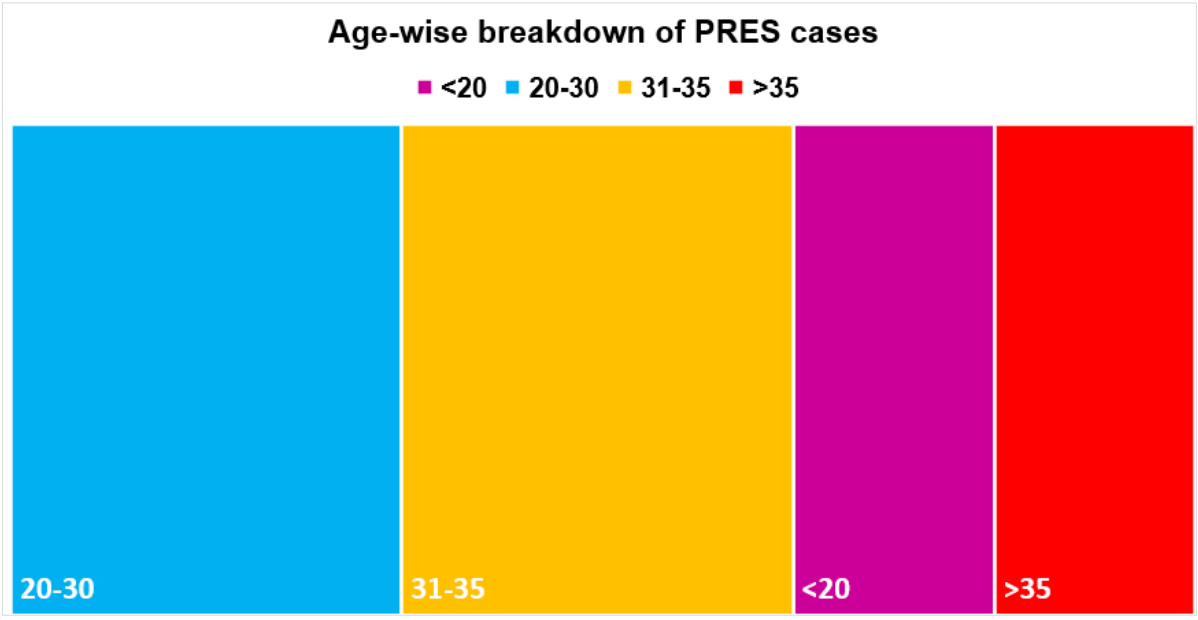

Figure 2: Age-wise breakdown of PRES cases

Table 4: PRES patients classification based on gravida status

Sl No	Gravida	Total number	Percentage
1	Primi	6	50
2	Multi	6	50

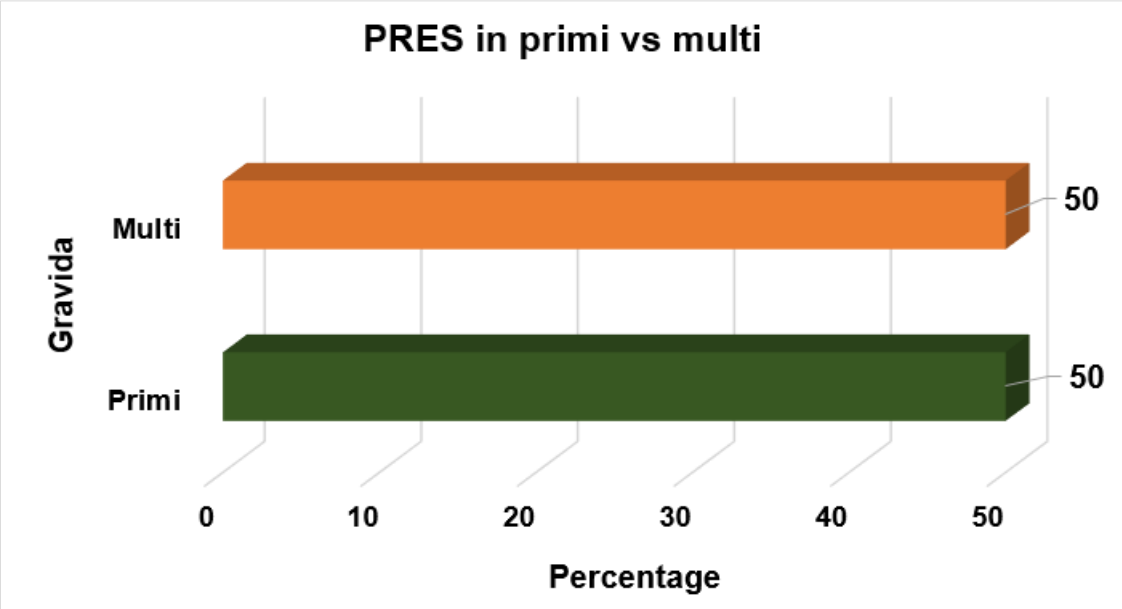

Figure 3: PRES in primi vs multi

Table 5: Clinical manifestations in PRES cases

Sl No	Clinical Presentations	Total number	Percentage
1	Headache	11	92
2	Vomiting+Nausea	4	33
3	Epigastric pain	1	8
4	Proteinuria	4	33
5	Blindness	5	42
6	Visual Disturbance	5	42
7	Previous pre-eclampsia	10	83.3

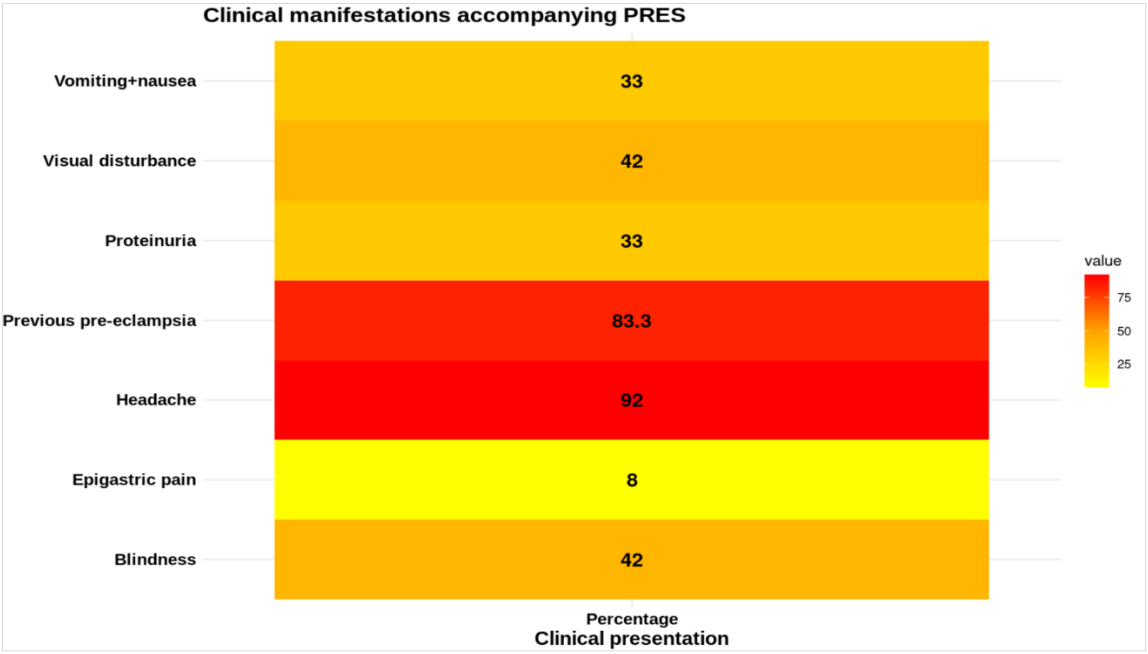

Figure 4: Clinical manifestations accompanying PRES

Table 6: Frequency of comorbidities in PRES patients

Sl No	Comorbidities	Total number	Percentage
1	Glasgow Coma Scale	2	17
2	Anaemia	4	33
3	Pulmonary edema	3	25
4	HELLP	1	8
5	Seizures	9	75

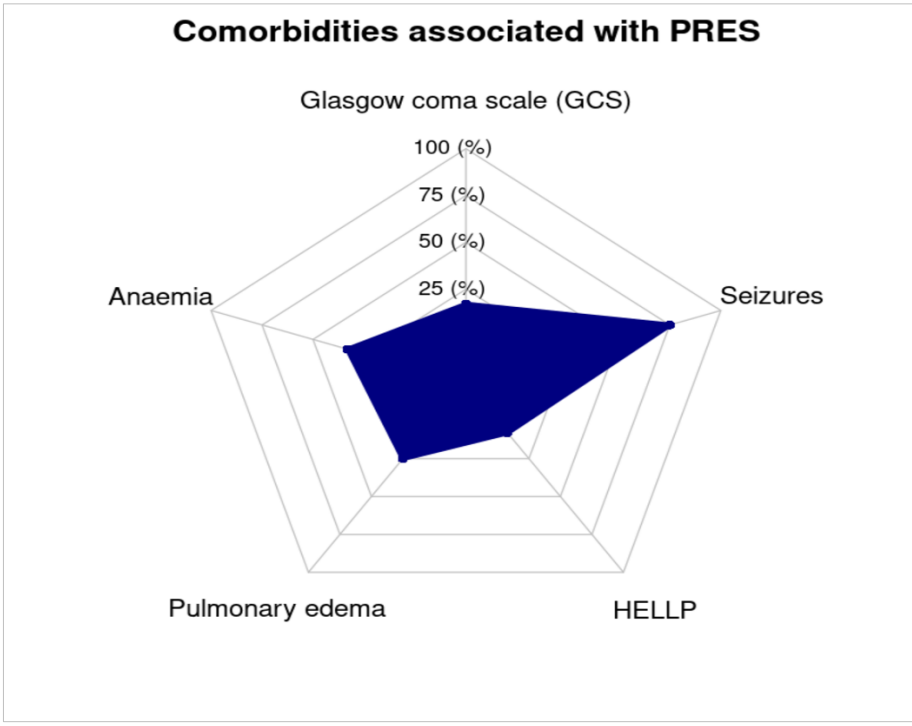

Figure 5: Comorbidities associated with PRES

Table 7: Clinical complaints and protocols of treatment in PRES patients

Sl No	Author	Complaints	Treatment
1	Maïga Youssoufa et al[10]	Blindness, headache, vomiting, seizure, proteinurea	On discharge: Carbamazepine 200 mg twice a day. In ICU: IV Nicardipine and $MgSO_4$
2	Ülkü Mete Ural et al[11]	Proteinurea, edema, blindness, headache	$MgSO_4$ 4g
3	Pooja Gupta Jain et al[12]	Blurring of vision, epigastric pain, seizures, vasogenic edema, headache	Labetalol 20 mg, 40 mg, $MgSO_4$ IV, Midazolam 600 mg, phenytoin 100 mg and antibiotics, LSCS was done and Labetalol 100 mg IV given
4	Makarim et al[13]	Loss of vision on both eyes, GDM, PIH, no neurological conditions, headache	Nifedipine 20 mg
5	Maasoumeh Mirazamoradi et al[14]	Seizures, altered mentation, visual loss, nausea and vomiting, edema, headache	$MgSO_4$
6	Antonio Sesar et al[15]	Seizures, loss of consciousness, bilateral loss of vision, headache, proteinurea, right sided facial nerve paresis, nausea	$MgSO_4$, Diazepam and antihypertensives (Case of LSCS 1st day)
7	Kaori Masai et al[16]	Nausea, difficulty in focusing, seizures, headache	In ICU: Levetiracetam 500 mg twice a day, edaravone 30mg once a day (one of the rarest and most unique condition, most delayed onset)
8	Majumder A et al[17]	DM, HTN, BA, disorientation, blurred vision, seizures, headache, nausea, vomiting, asked to follow-up regularly for lupus anticoagulant factor, was taking NSAIDs	Broad spectrum antibiotics, phenytoin, dexamethasone
9	Niruby Rasendrakumar[18]	GDM, urticaria, seizures, tendon reflexes brisk, visual disturbances, headche	Levetiracetam and Labetalol twice a day, $MgSO_4$, Midazolam 2 mg
10	Suman Nishad et al[19]	Seizures, headache	Labetalol, Levetiracetam
11	Manmin Zhu et al[20]	Loss of consciousness, twitching, no h/o eclampsia, decreased visual acuity in both eyes, slightly elevated protein, headache	Amlodipine besylate for HTN, Sodium valproate, Oxcarbazepine
12	Anh Dinh Bao Vuong et al[21]	Altered mental health,pulmonary edema, visual disturbance, seizures,	Micardipine 10 mg/10 ml and IV infusion $MgSO_4$, antibiotics (after 4 days from onset), Levetiracetam 500 mg and Haloperidol 2 mg twice a day (Diagnosed with severe pre-eclampsia and HELLP syndrome. Corticosteroids given for lung maturity (emergency LSCS).

GDM: gestational diabetes mellitus; PIH: pregnancy induced hypertension; DM: diabetes mellitus; BA: bronchial asthma; $MgSO_4$: magnesium sulphate; NSAID: non-steroidal anti-inflammatory drug

Table 8: Approaches of treatment in PRES patients

Sl No	Treatment	Total number	Percentage
1	$MgSO_4$	6	50
2	$MgSO_4$+Phenytoin	3	25

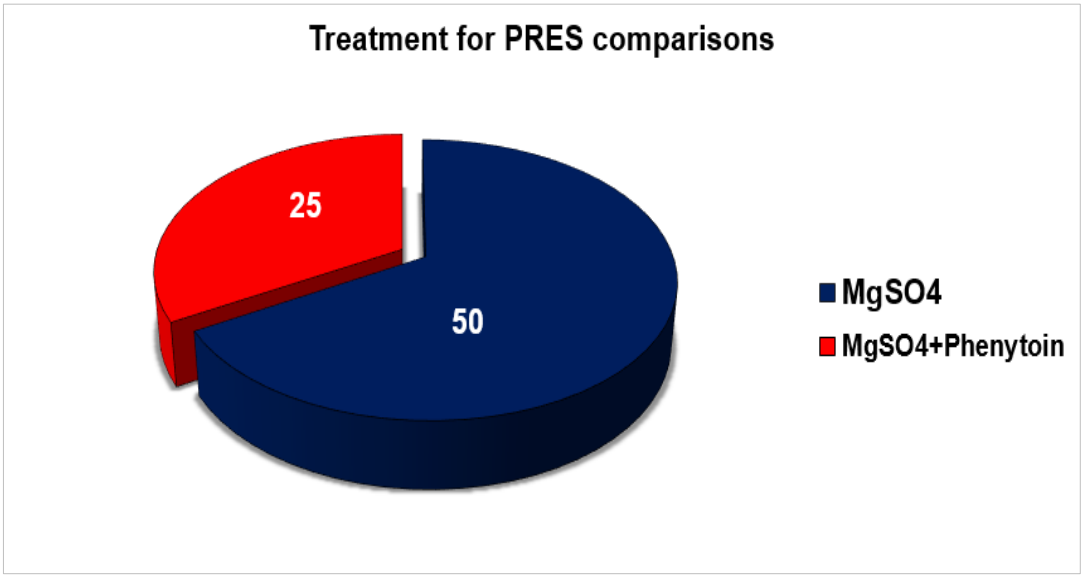

Figure 6: Treatment for PRES comparisons

Discussion

Eclampsia is a common etiology for PRES [22]. The incidence of PRES is 75% in eclampsia [23]. Albeit, the exact etiology is unknown [24]. However, eclampsia is an important etiology in PRES [25]. The prevalence of PRES incidence was noted mostly in the age-group of 20-35 years in contrast to a study [26]. The incidence of developing PRES increases with decreasing age [27]. The most common clinical feature was noted as headache. Similar reports have been given in other studies too. It is also supported by another study [28]. Most often headache is not considered as a significant feature in post-partum cases, it is taken very lightly. Headache is considered as a hallmark in the diagnoses of PRES which can be easily missed [29]. HTN is noted in PRES in most of the cases with a prevalence of 83.3%. This is supported by another study [30]. Proteinuria was noted in 4 patients (33%) in our study. Anyway proteinuria is not an essential diagnosis for PRES. So also in the case of eclampsia and pre-eclampsia [31]. Another feature is blindness which was noted as a prevalence of 42% due to cortical disorder [32]. Here the people reacted to light and fundus examination was normal with no papillary edema. But it is reversible in the case of PRES after timely treatment. Seizure is characterized by tonic and clonic contractions [33]. Hyper-reflexia was noted in 75% of cases but the Babinski's sign turned out to be negative. The most important differential diagnoses of PRES are cerebral venous sinus thrombosis [34], cerebrovascular disease due to infection, epilepsy [35], patient on chemotherapeutic drugs, patients with transplanted organs taking immunosuppressive drugs [36]. A rare case of PRES developing on the 71st day after the normal vaginal delivery was also noted (Kaori et al). Brain MRI showed high signals on T2 weighted images and FLAIR in PRES cases [37]. MR angiography shows focal vasoconstriction and vasodilation along with a string of beads appearance in posterior cerebral arteries bilaterally. But in the absence of MRI angiogram only MRI FLAIR can be taken into consideration, CT may be negative for diagnosis of PRES [38]. CT may in fact not be of diagnostic value and the condition of PRES may be missed [39]. Treatment with $MgSO_4$ and combination of drugs were used in our study [40]. An interesting study shows hypomagnesemia is a cause of PRES. Hence, treatment with $MgSO_4$ is done [41].

Conclusion

The risk factors and the symptoms associated with eclampsia are well known among the obstetricians, but the development of PRES in eclamptic patients should be kept in mind. The most common symptom is headache among the patients. An early MRI will help in instant diagnosis of PRES. This initiative may be helpful for researchers in the future to study further in preventing PRES. So our study may help in an initial step to future discovery.

A multidisciplinary team strategy should be sought in healthcare for other facilities like ophthalmologist, cardiologist, anesthetist and neuro physicians. A well-established ICU setup with the essential drugs and early diagnosis of PRES awareness should be there among paramedics and the public. A humble request for the junior young physicians in emergency department and the paramedics that they should be aware of the patients coming with headache, nausea and vomiting to exclude PRES and not to send home the patients who have delivered. There should be a lurking doubt and a healthy skepticism in the mind when the patients come even after 10 weeks of delivery complaining of headache. It is mandatory and vigilance is the key for early diagnosis and prevention of maternal mortality.

To sum up, this review highlights the need for the medical personnel to explore PRES in postpartum eclampsia patients and to initiate an early diagnosis and a multidisciplinary management approach that may improve maternal outcomes and reduce risk of persistent neurologic injury.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation:
Artificial intelligence(AI) was not used in writing assistance or manipulation of images.

Limitation

The period of study taken is short with a period of 12 years. The study has omitted the antepartum and intra partum obstetric patient. The non-obstetric patients like PRES with SLE, chemotherapy, drugs and other causes are not dealt. The study sample is also small. Paediatric patients are not included.

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Conflicts of interests

The authors report no conflict of interest.

Author contributions

Conceptualization and methodology, A.T.C., S.S., and J.K.S.; Formal analysis, B.T.R., and J.K.S..; Visualization and writing – original draft J.K.S., B.T.R., S.S.; Writing - review and editing, A.T.C., S.S., J.K.S., B.T.R., and J.H. All authors have read and agreed to the final version of the manuscript.

Ethical approval

Not Required

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