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Original Research Article

ROLE OF MAGNETIC RESONANCE IMAGING IN THE DIAGNOSIS OF POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME AND ITS OUTCOME IN ECLAMPSIA PATIENT – A PROSPECTIVE STUDY

ABSTRACT

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Aim: Our study's objective is to assess the degree and type of brain edema, incidence of atypical and typical regions of involvement and unusual imaging manifestations as well as to assess the neuro imaging abnormalities in eclampsia patients with PRES.

Material and Method: This is a referral hospital-based prospective study of 50 consecutive cases of eclampsia who were subjected to MRI brain. All 50 women with eclampsia were treated with routine principles of management of eclampsia.

Results: We observed a combined pattern of typical and atypical PRES compared to individual findings. Among the atypical patterns of PRES, the holohemispheric atypical pattern was observed to be highly significant compared to other atypical patterns of PRES. 36% of patients were observed with postpartum eclampsia which is most common in duration of less than 48 hours. During follow-up, reversible eclampsia with PRES was highly observed compared to irreversible.

Conclusion: Magnetic resonance imaging is a useful imaging technique that improves the diagnosis as well as differentiates the types of PRES .MRI could help early detection of PRES in eclampsia patients for better prognosis and overall improvement of maternal and child health.

Keywords: Posterior reversible encephalopathy syndrome(PRES), eclampsia, Magnetic resonance imaging.

1. INTRODUCTION

Posterior reversible encephalopathy syndrome is a clinicoradiological syndrome characterized by symptoms including a headache, seizures, altered consciousness, visual disturbances, and radiological features of white matter vasogenic edema. It was first

described in 1996 by Hinchey et al.[1] It is described in association with various conditions like hypertension, pre-eclampsia, eclampsia, infections, autoimmune diseases, and cancers.[2] Both clinical and radiological characteristics are usually reversible.[3-5] The authors have studied the role of MRI in the evaluation of PRES syndrome and its outcome.

The posterior reversible encephalopathy syndrome (PRES) is the most common neuroimaging sign of eclampsia and preeclampsia.[6,7] With its distinctive neuroimaging findings of vasogenic edema involving the posterior circulation, the posterior reversible encephalopathy syndrome (PRES) is a clinically distinct entity that manifests with neurologic signs and symptoms (headache, altered consciousness, visual abnormalities, and seizures). In 1996, Hinchey et al[8] were the first to report a link between eclampsia and PRES. Three of the 15 individuals in this first group who had PRES also developed eclampsia. Immunosuppressant medications and hypertensive encephalopathy were additional causes of PRES.

Preeclampsia-eclampsia with PRES currently lacks a clear pathophysiological mechanism, which is still debatable.[9-11] The extent and pattern of brain edema in eclampsia and preeclampsia patients with PRES may differ, and there may also be a "threshold" trigger that causes eclampsia, but this has not yet been established.[12,13]

Cranial conventional magnetic resonance imaging (MRI) is the preferred imaging modality for preeclampsia-eclampsia patients with PRES.[6].**Gao et al.** used conventional MRI sequences to score the extent of brain edema in PRES patients and found that the score was significantly correlated with serum levels of lactate dehydrogenase.[9] Diffusion-weighted imaging (DWI) is another commonly performed MRI sequence and is highly sensitive for distinguishing between cytotoxic and vasogenic edema.[6] Recent MRI studies have shown that DWI can reflect the changes in pathophysiology regarding PRES patients in the ictal or peri-ictal phase of epilepsy.[12] The purpose of this prospective research was to compare the extent and nature of brain edema in eclampsia patients with PRES based on the MRI characteristics

2. MATERIAL AND METHODS

A prospective study is conducted in the Department of Radiodiagnosis, super specialty block at our institute to describe the features of PRES in eclampsia symptomatic females based on MRI.50 patients with clinical suspicion of eclampsia who will be referred to our department were subjected to MRI.

All eclamptic women who are admitted to our tertiary care referral hospital for neuroimaging studies are thoroughly analyzed. Patients with diagnoses of eclampsia are used to select cases for follow-up. Women who undergo neuroimaging are recruited for the study, and the necessary records are gathered. Most of these scans are performed on new admissions within the first 24 hours. Patients with eclampsia who have undergone cranial imaging were identified, analyzed, and relevant data were retrieved from their medical records. No additional information is gathered on eclamptic patients if neuroimaging is not performed on them. Patients are enrolled in the study once they have given their informed consent. Thereafter every patient will get an MRI scan done. Abnormalities in neuroimaging are evaluated in these patients. The patient's informed consent was taken before the examination, the procedure explained, screened for contraindications, and taken into the MRI machine.

MRI was done by using "Philips -Ingenia (1.5 Tesla dStream) with brain coil". The data from the MRI brain studies of 50 patients performed between November 2020 to July 2022.

Inclusion Criteria

- History of eclampsia.
- Initial MRI showed cortical or subcortical FLAIR and T2-weighted hyper-intensity with posterior predominance that resolved or significantly improved on follow-up MRI or CT.
- Initial MRI showed cortical or subcortical FLAIR or T2-weighted hyperintensity with posterior predominance in a parietooccipital distribution typical of PRES but lacking repeat imaging; these cases without repeat imaging were to be confirmed PRES only if the patient had a complete return to baseline neurologic status. In addition, for these patients to be included, they had to have taken a drug or had a condition known to cause PRES that was treated or removed before the symptoms went away completely, and the clinician had to agree that PRES caused the symptoms.
- Initial MRI showed T2-weighted or FLAIR hyperintensity in the brainstem, basal ganglia, or subcortical or cortical frontal regions without posterior predominance (atypical distribution), and the imaging findings resolved or significantly improved on follow-up MRI in the setting of a cause previously attributed to PRES.
- Cases were included irrespective of age.

Exclusion Criteria

- Patients not giving consent.
- Contraindications to MRI study like patients with pacemakers, metallic implants, and aneurysmal clips.
- Claustrophobia or anxiety disorder aggravated by MRI.
- Cases lacking both clinical and imaging findings are excluded.

Imaging Parameters: Following sequences were used

1. AxialT1W
2. Axial/sagittalandcoronalT2Wsequences
3. Axial FLAIR
4. Axial Diffusion weighted sequences – DWI and apparent diffusion coefficient –ADC
5. Axial susceptibility imaging sequences.

The field of view is 14-16 cm, with a slice thickness 2-3 mm and a matrix of 512 x 512.

Ethical Consideration: Approval was taken from the ethical committee of Gajra Raja Medical College for obtaining the information regarding patients admitted to Kamla raja hospital Gwalior. The approval number assigned to our study is 135/IEC-GRMC/2020.

Statistical analysis

Analysis was done after entering the data in Microsoft Excel and evaluating it using the SPSS Statistics software version 17.0 (IBM, Armonk, NY .and analysed using percentages and mean.

The association between the frequency distribution of PRES syndrome in primigravida and multigravida and its outcome was calculated using the chi-squared test. A p-value <0.05 was considered statistically significant.

During the follow-up of our study population, we observed that reversible PRES (84%) was significantly more observed compared to irreversible PRES (16%). ($p = <0.0001$).

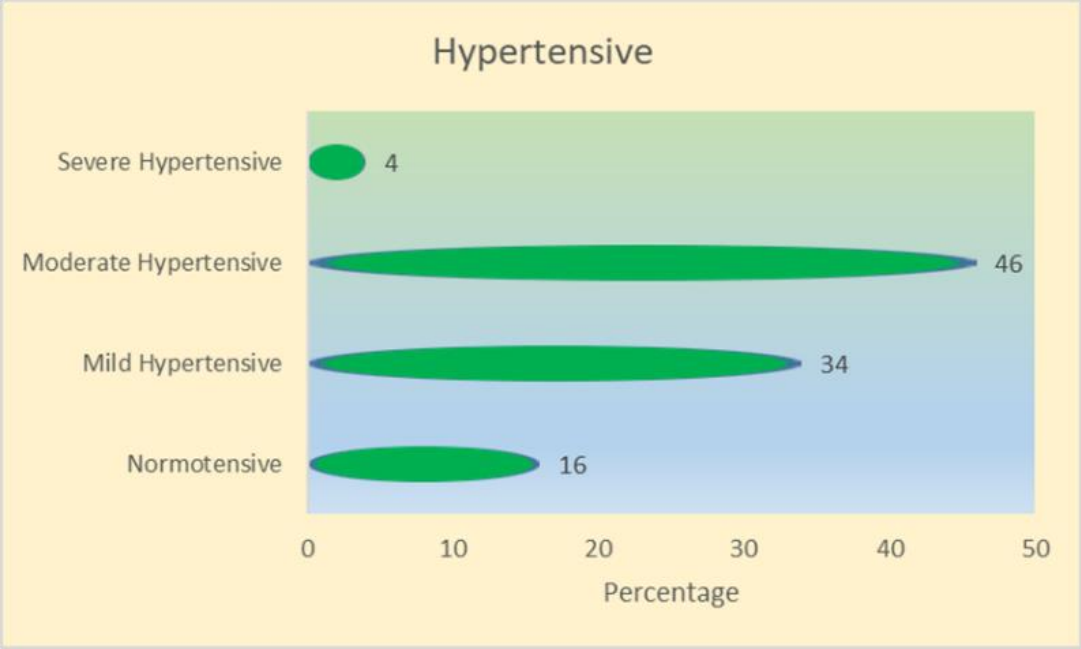
3. OBSERVATION AND RESULTS

We registered 50 patients, all of whom had PRES with eclampsia. These patients were prospectively enrolled between November 2020 and July 2022 at our institute. We selected 50 patients based on exclusion as well as inclusion standards. The overall mean age of the study was 27.42 ± 5.42 years. Among 50 patients, the highest percentage was observed in the age group of 21–25 years. The lowest percentage was observed in the age group of 15-20 years. In the age group of 26–30, 31–35, and 36– 40, the age groups were observed as follows: 28%, 18%, and 10%.

Out of 50 patients, 33 patients who were diagnosed with PRES syndrome, were primigravida (66%) and 17 were multigravida (34%) hence PRES was significantly more frequently observed in primigravida as compared to multigravida. (p = 0.0015)

In relation to gestation age, the percentage of PRES was high (44%) at the gestation age of 29-36 weeks as compared with <29 weeks and >36 weeks.

Five groups of eclampsia patients were identified: 1) Normotensive (16%) 2) High blood pressure (84%) 3) Mild hypertension (34%).4) Moderate Hypertensive (46%) 5. Serious hypertension (4%). We were highly observed as hypertensive (84%) compared to others in our study group mentioned in Graph 1.



Graph 1 :- Depicts types of Eclampsia

Symptoms:

We observed the following number of symptoms in eclampsia with PRES patients.1) Headache: 25 (50%)2) Altered Sodium: 16(32%) 3) Seizures: 45 (90%) 4) Visual Disturbances: 19 (38%) and 5)Neurological deficit: 8(16%). Seizures had the highest percentage of symptoms, while neurological deficits had the lowest number of symptoms.

Antepartum and Postpartum:

The average antepartum was observed at 235.22 ± 26.21. Among postpartum, 22%

observed at <48 hours, 8% observed at 48 hours to 7 days, and 6% observed at >7 days.

Types of PRES:

Among PRES, 24% had typical PRES, 36% had Atypical PRES and 40% had a combined pattern of PRES observed in our study. In our study population, atypical patterns of PRES were significantly more highly distributed in the Superior frontal sulcus pattern (18%), then the holohemispheric watershed pattern (14%), and then after other types of PRES(4%) in our data. We observed 3 types of atypical patterns. 1) Frontal sulcus pattern superior (18%) 2) the holohemispheric watershed pattern (12%) and 3) others (6%).

During the follow-up of our study population, we observed that reversible PRES (84%) was significantly more observed compared to irreversible PRES (16%). (p = <0.0001) mention in table no. 4 and graph 2

Table 1: Relation of typical pattern with age and blood pressure

Parietooccipital Pattern	Typical MRI (Absent)	Typical MRI (present)	p-value
Age	28.29±5.69	24.67±3.37	0.0420
SBP	165.68±2726	165.50±29.53	0.9840
DBP	95.84±12.26	94.83±13.06	0.8080

According to our data analysis, we observed a mean age significantly higher in Atypical PRES than in the typical pattern of PRES. (p = 0.011; 95% CI mean difference = 3.623 (0.894-6.352). Systolic and diastolic blood pressure were not significantly observed between Atypical PRES and typical PRES.

Table 2: Relation of atypical pattern with age and blood pressure

Holohemispheric pattern	Atypical MRI (Absent)	Atypical MRI (present)	p-value
Age	26.79±5.20	31.29±5.53	0.0410
SBP	163.30±27.20	180±26.88	0.1380
DBP	94.51±12.34	102.29±10.74	0.1230

Based on the results of an independent t-test, we saw that age was a very important factor in holohemispheric pattern type of Atypical PRES compared to other groups. Systolic and diastolic blood pressure was not significantly observed between the holohemispheric pattern type of Atypical PRES and other types of PRES.

Table 3: Logistic model

		B	S.E.	Wald	df	Sig.	Exp(B)
Step1[a]	Age	0.118	0.122	0.942	1	0.332	1.125
	SBP	0.008	0.076	0.011	1	0.917	1.008
	DBP	0.134	0.159	0.711	1	0.399	1.144
	No-hy			1.978	3	0.577	
	No-hy(1)	-5.006	3.954	1.603	1	0.205	0.007
	No-hy(2)	-6.241	6.384	0.956	1	0.328	0.002
	No-hy(3)	-29.632	26597.021	0.000	1	0.999	0.000
	Headache(1)	1.673	1.772	0.892	1	0.345	5.328
	Altered	1.264	1.271	0.989	1	0.320	3.539

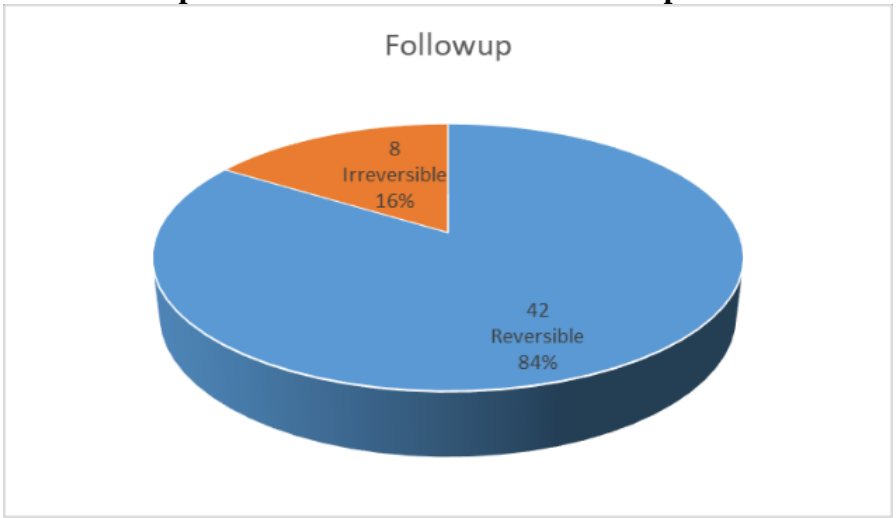
	sensorium(1)						
	Visual disturbance(1)	0.377	1.471	0.066	1	0.798	1.457
	Neurological deficit(1)	0.812	1.353	0.360	1	0.548	2.253
	Primi/Multi(1)	0.461	1.315	0.123	1	0.726	1.585
	Constant	-16.567	13.335	1.543	1	0.214	0.000
a.Variable(s)enteredonstep1:Age,SBP,DBP,No-hy,Headache,Altered sensorium,Visualdisturbance,Neurologicaldeficit,Primi0/Multi1.							

According to logistic regression, reversible or irreversible is not rectified by the factor associated with them. In our model, we included several parameters, such as age, blood pressure, normotensive, hypertensive, mild hypertensive, severe hypertensive, headache, altered sensorium, visual disturbance, neurological deficit, and added gravidae. So, we could not predict based on that.

Table 4: Outcome-wise distribution of patients

	Frequency	**Percentage**	**p-value**
Reversible	42	84	<0.0001
Irreversible	8	16	

Graph 2: Outcome-wise distribution of patients



Case no-1

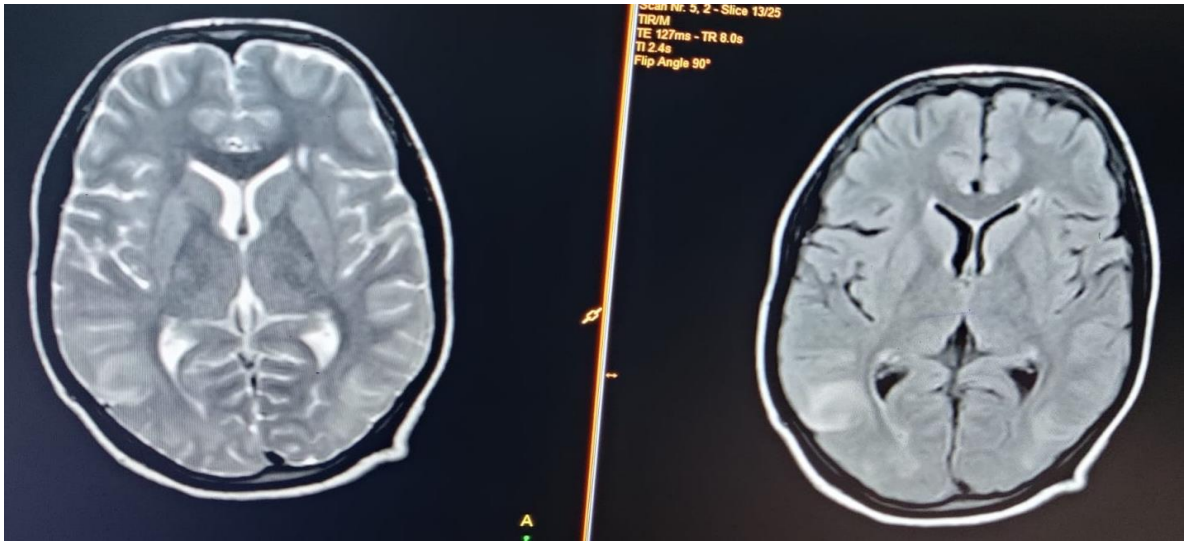


Figure 1 - Axial T2-WI and FLAIR images showing confluent and focal area of T2/FLAIR hyperintensities noted in bilateral parietooccipital lobes-Typical PRES.

Case-2

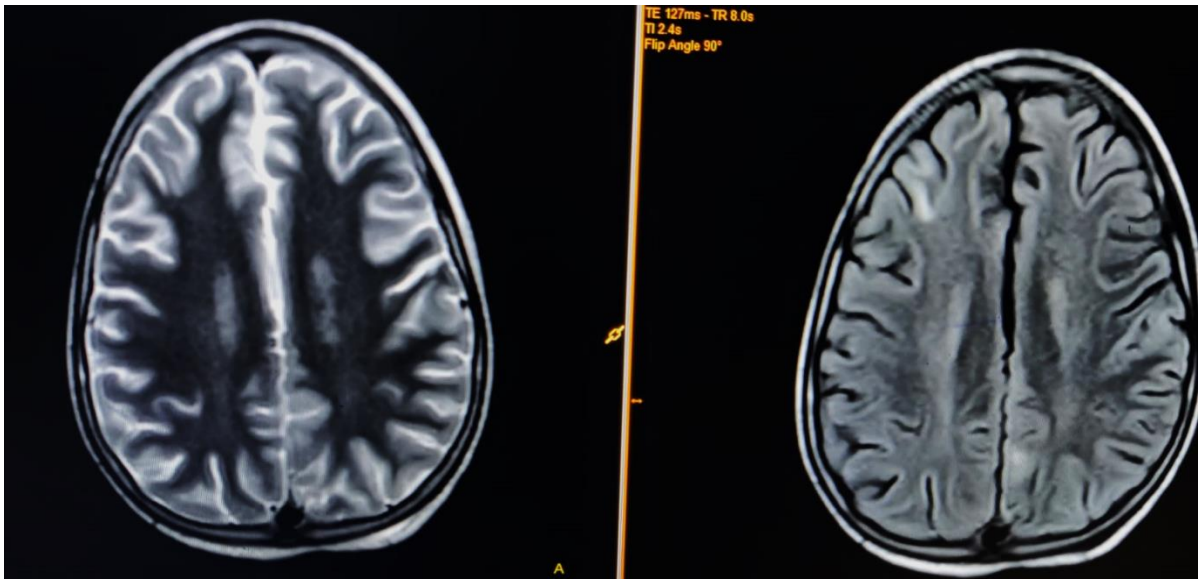


Figure 2 : Axial T2-WI and FLAIR images showing focal gyral FLAIR hyperintensity noted in right frontal region- superior frontal sulcus pattern- Atypical PRES

Case - 3

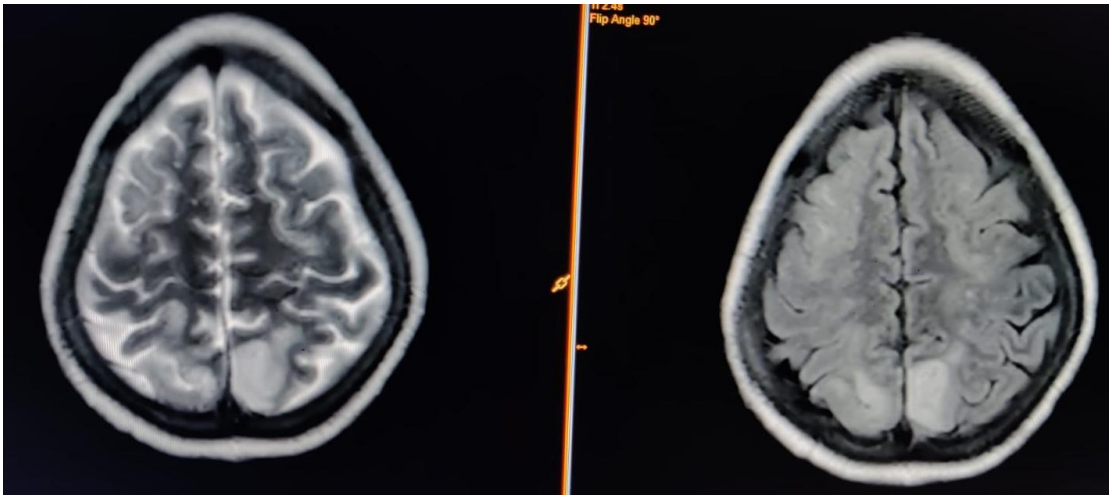


Figure 3 : Axial T2-WI and FLAIR images showing multiple focal area of T2/FLAIR hyperintensities noted in bilateral high parietal lobes, bilateral centrum semiovale, bilateral corona radiata, right internal capsule, left putamen, bilateral white matter cerebral hemispheres and splenium of corpus callosum- Holohemispheric watershed pattern-Atypical PRES

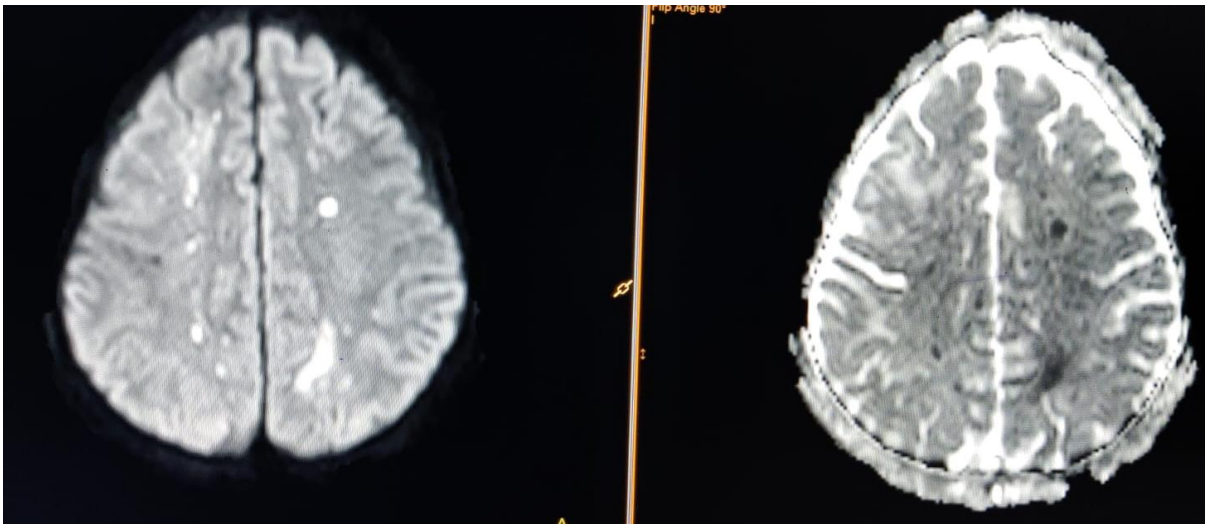


Figure 4: Axial DWI and ADC images showing above mentioned T2/FLAIR hyper intensities showing DWI restriction.

Case-4

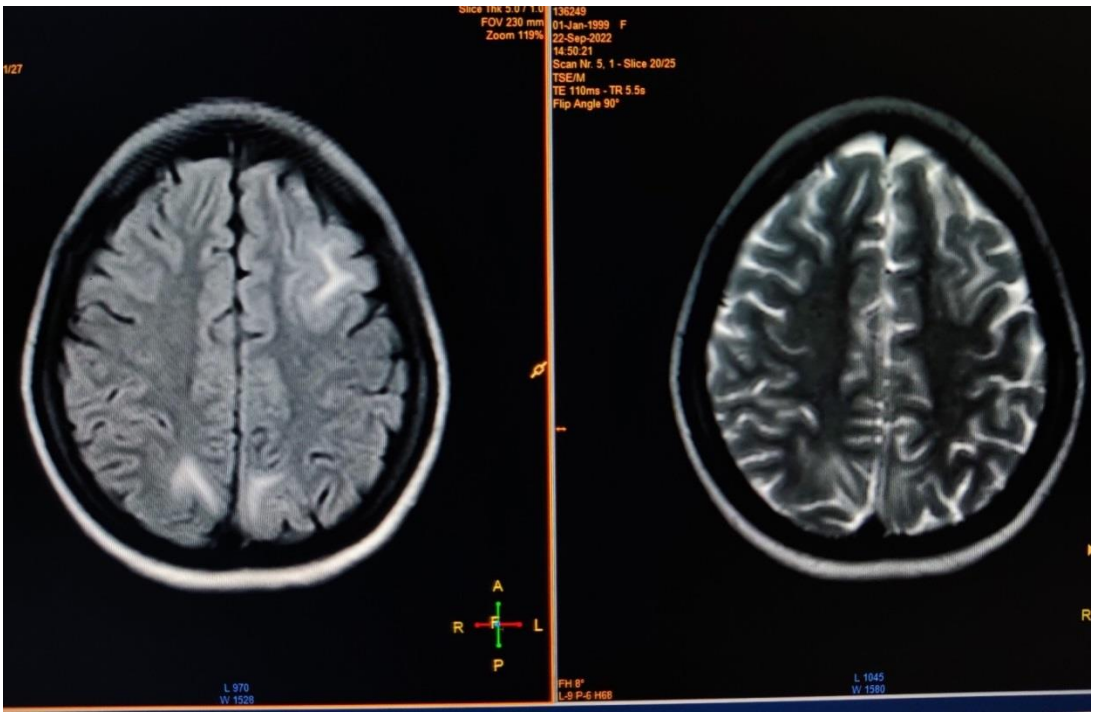


Figure 5 : Axial FLAIR and T2-WI images showing T2/FLAIR hyperintensities noted in subcortical white matter of bilateral frontal and bilateral pariteal lobes - combination PRES.

Case-5

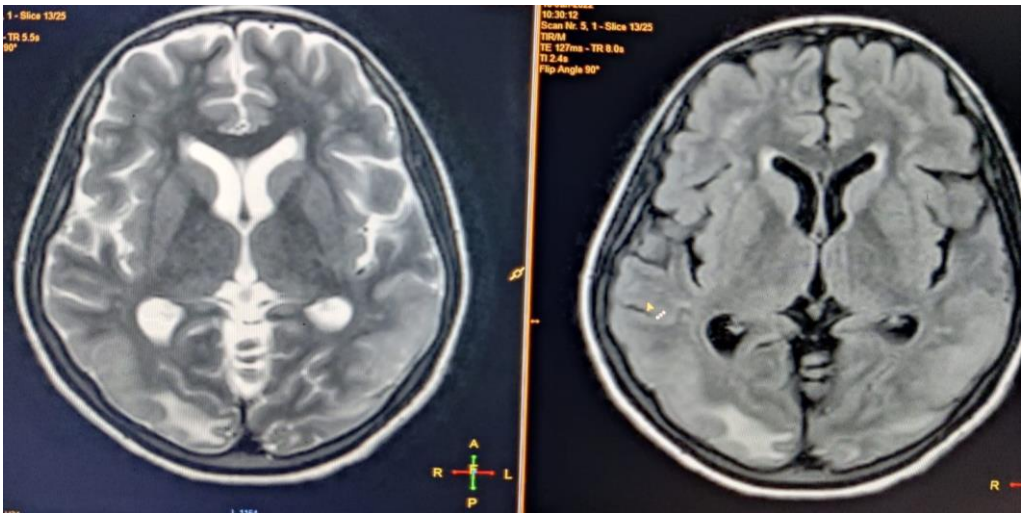


Figure 6 : Axial T2-WI and FLAIR images showing multiple confluent areas of T2/FLAIR hyperintensities noted in bilateral parietal lobes, bilateral frontal lobes and bilateral occipital lobes-combination PRES

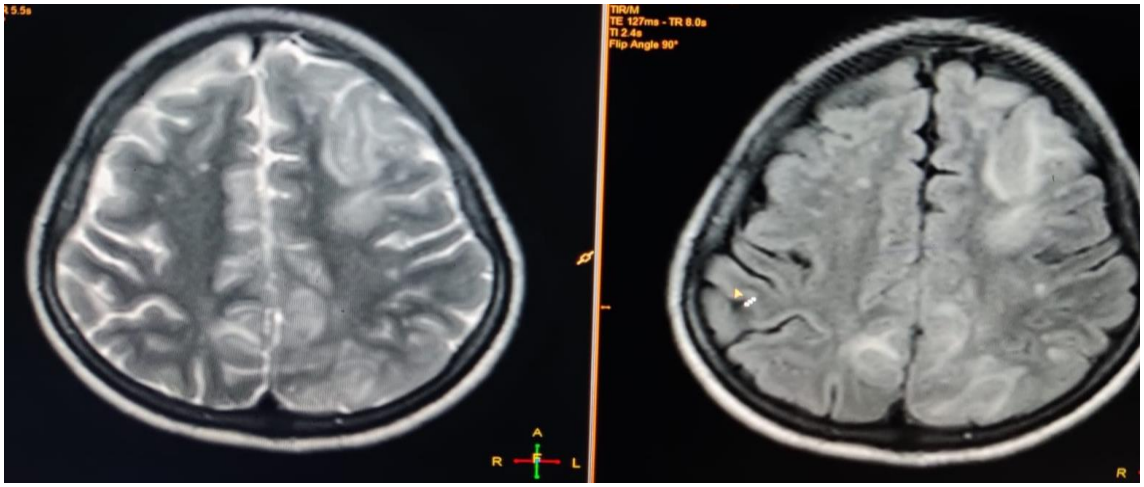


Figure 7 : Axial T2-WI and FLAIR images showing T2/FLAIR hyperintensities noted in bilateral frontal and bilateral parietal lobes.

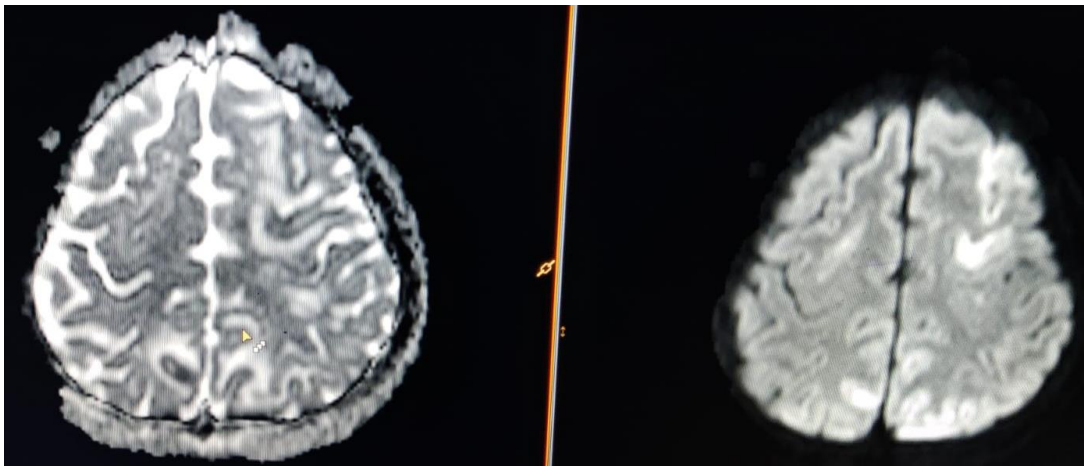


Figure 8: Axial DWI and ADC images showing above mentioned T2/FLAIR hyperintensities showing DWI restriction

4. DISCUSSION

Preeclampsia is a systemic syndrome in pregnancy characterized by hypertension and proteinuria. The diagnosis of eclampsia is established if a seizure occurs together with preeclampsia which cannot be explained by other causes. The incidence of eclampsia in developed countries averages 1 in 2000 to 3000 deliveries.[14,15]

PRES with eclampsia is a rare condition and is associated with headache, seizure, altered mentation, and visual disturbances. Posterior Reversible Encephalopathy Syndrome (PRES) is also known as reversible posterior leukoencephalopathy syndrome.[16-19] PRES is associated with an abrupt increase in blood pressure. It can present with focal neurologic deficits, mimicking a stroke, and can also present atypically. The major causes of this syndrome are severe hypertension, preeclampsia, eclampsia, renal failure, vasculitis, TTP, immunosuppressive treatment, sepsis, and hyperammonaemia. The pathogenesis of this syndrome is the inability of the brain circulation to auto-regulate, in response to acute changes in blood pressure. Vasogenic edema occurs due to disruption of the blood-brain

barrier. When unrecognized, irreversible cytotoxic edema may occur.[20]

PRES are observed during pregnancy and late in the postpartum period after an uneventful pregnancy. It is usually associated with late eclampsia and a high rate of cortical visual loss in these patients.[21] Visual complications in preeclampsia are hypertensive encephalopathy, retinal detachment, and cortical blindness while visual abnormalities in PRES are: blurred vision, visual neglect, homonymous hemianopia, visual hallucinations, and cortical blindness.[22]

In our study among 50 women, PRES with eclampsia candidates were selected according to the inclusion-exclusion criteria, and MRI brain was done. The reports were analyzed and studied. Among 50 women, 66% have primigravidae and 34% have multigravidae.

In our study, PRES has 84% hypertensive whereas 16% normotensive patients. Hypertensive is categorized into 3 parts: 1) Mild Hypertensive 2) Moderate hypertensive and 3) Severe hypertensive. Among all hypertensive patients, Moderate hypertensive (46%) was highly observed.

Sardesai et al., (2019)[23] observed the most common age group 20-25 years in PRES with eclampsia. Our study also represents similar results to her study. The most common age group was 21-25 years in our study. 38% of the patients fall between 21-25 years of age whereas least percentage was observed in the age group of <21 and 36-40 age group. The mean age of the study population was 27.42 ± 5.42 years.

According to our study, MRI pattern of PRES observed mainly 2 types: 1) Typical pattern of PRES (24%) 2) Atypical pattern of PRES (36%), and combined patterns (40%). In the typical pattern of PRES, all the patients have parieto-occipital lobes lesions (100%) which is similar to the results of Goyal G et al.,(2022). Our study was mainly focused on the atypical pattern of PRES. So, Atypical PRES of MRI observed the following patterns: superiorfrontal sulcus (50%), holohemispheric pattern(33.33%), and other (16.67%).

During follow-up of our study, 91.67% of patients having reversible PRES who have before the typical pattern of PRES. Among Atypical PRES patterns, the superior frontal sulcus and holohemispheric have 100% reversible PRES whereas other remaining atypical patterns of PRES have 100% Irreversible PRES. The combined pattern of PRES has 20% Irreversible PRES and 80 % reversible PRES.

Goyal G et al., (2022)[24] present PRES with eclampsia having symptoms such as seizures 66.6%, headache 40%, and visual disturbance 20%and altered sensorium 53.3% whereas our study represents PRES with eclampsia patients having symptoms as followings: seizures 45 (90%), headache 25(50%), visual disturbance 19 (38%), altered sensorium 16 (32%) and neurological deficit 8 (16%). These above-mentioned data were similar to the study of be M Balgi et al., (2015)[25] and Mayama Michinori et al.,(2016).[26]

According to Sardesai S et al., (2019)[23] study, 75.66% antepartum and 24.54% postpartum types of eclampsia were observed. We also observed 64% antepartum eclampsia and 34% postpartum eclampsia. Highly antepartum eclampsia (68.75%) was observed in pregnant women who have 29-36 weeks of gestational age whereas postpartum eclampsia (61.11%) was observed during <48 hours.

Study	Eclampsia Patients	Post-partum (%)
Douglas and Redman,1994[27]	383	5
Conde-Agudelo and Kafury-Goeta,1998[28]	164	12
Katzetal,2000[29]	53	6

Mattar and Sibai,2000[30]	399	17
Chames et al,2002[31]	89	26
Chenetal,2003[32]	62	3
Sardesai et al,2019[23]	110	24.54
Present study	50	34

5. CONCLUSION

Posterior reversible encephalopathy syndrome (PRES) is a condition that is commonly encountered in clinical practice, with prompt recognition and intervention to remove precipitating factors serving to optimize patient outcomes and reverse symptoms as well as imaging changes. It is of particular importance not to exclude PRES as a possible diagnosis when we have the appropriate clinical presentation which is not accompanied by the typical radiological findings since this clinical-radiological syndrome can often be manifested with atypical MRI findings, hence the recognition of atypical imaging manifestation of PRES is important to avoid delays in diagnosis and treatment as is the identification of complicating factors which may adversely affect patient prognosis. The success of the therapy lies primarily in recognizing these findings. Based on our findings, we may conclude that magnetic resonance imaging is a useful imaging technique that improves the diagnosis as well as differentiates the types of PRES.

We thank our senior consultants and statistician who provided a huge effort to contribute to this very interesting Research Topic. We are also grateful to the reviewers who did not count their time in allowing the production of these quality articles. We hope that this research topic might be a real step forward in the understanding of this intriguing and exciting syndrome.

6. REFERENCES

1. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A, et al. A reversible posterior leukoencephalopathy syndrome. N Engl J Med. 1996;334:494-500.
2. Lee VH, Wijdicks EF, Manno EM, Rabintein AA. Clinical spectrum of reversible posterior leukoencephalopathy syndrome. Arc Neurol. 2008;65(2):205-10.
3. Bartynski WS. Posterior reversible encephalopathy syndrome, part 1: fundamental imaging and clinical features. AJNR Am J Neuroradiol. 2008;29(6):1036-42.
4. Garg RK. Posterior leukoencephalopathy syndrome. Postgrad Med J. 2001; 77:24-28.
5. Kastrup O, Maschke M, Wanke I, Diener HC. Posterior reversible encephalopathy syndrome due to severe hypercalcemia. J Neurol. 2002;249:1563-6.
6. Garg RK, Kumar N, Malhotra HS. Posterior reversible encephalopathy syndrome in eclampsia. Neurol India. 2018;66:1316–1323.
7. Demirtas O, Gelal F, Vidinli BD, Demirtas LO, Uluc E, Baloglu A. Cranial MR imaging with clinical correlation in preeclampsia and eclampsia. Diagn Interv Radiol. 2005;11:189–194.
8. Hinchey J, Chaves C, Appignani B, et al. A reversible posterior leukoencephalopathy syndrome. N Engl J Med. 1996;334(8):494- 500.
9. Gao B, Liu FL, Zhao B. Association of degree and type of edema in posterior reversible encephalopathy syndrome with serum lactate dehydrogenase level: initial experience. Eur J Radiol. 2012;81: 2844–2847.

10. Ollivier M, Bertrand A, Clarencon F, Gerber S, Deltour S, Domont F, et al. Neuroimaging features in posterior reversible encephalopathy syndrome: a pictorial review. J Neurol Sci. 2017;373:188–200.
11. Mayama M, Uno K, Tano S, Yoshihara M, Ukai M, Kishigami Y, et al. Incidence of posterior reversible encephalopathy syndrome in eclamptic and patients with preeclampsia with neurologic symptoms. Am J Obstet Gynecol. 2016;215:239 e1–239 e5.
12. Wakisaka K, Morioka T, Shimogawa T, Murao K, Kanazawa Y, Hagiwara N, et al. Epileptic Ictal Hyperperfusion on Arterial Spin Labeling Perfusion and Diffusion-Weighted Magnetic Resonance Images in Posterior Reversible Encephalopathy Syndrome. J Stroke Cerebrovasc Dis. 2016;25:228–237.
13. Xiaobo F, Yanling L, Dunjin C, Fang H, Jia C, Yuhua Z, et al.Effect of blood pressure on reversible posterior leukoencephalopathy syndrome in pre-eclampsia or eclampsia. Hypertens Res. 2018;41:112–117.
14. Wagner, Steven J., et al. ―Posterior reversible encephalopathy syndrome and eclampsia: Pressing the case for more aggressive blood pressure control.‖ Mayo Clinic Proceedings. Vol. 86. No. 9. Elsevier, 2011.
15. Zhang, Lihong, et al. ―Late postpartum eclampsia complicated with posterior reversible encephalopathy syndrome: A case report and a literature review.‖ Quantitative imaging in medicine and surgery 5.6 (2015): 909.
16. Vinod SP, Dhamangaonkar BR, Pattanshetti RC, Patil MM. Posterior Reversible Encephalopathy Syndrome in Early Postpartum Women: A Case Report. Journal of Clinical and Diagnostic Research. 2014;8(4):RD01-2.
17. Justin B, Michelle YO, Kedra W, Amanda AR, Rachael M, Majid K, et al. Posterior reversible encephalopathy syndrome in 46 of 47 patients with eclampsia. Am J Obstet Gynecol. 2013;208:468.e1-6.
18. Anjan T, Ankur S, Rakesh K. Posterior reversible encephalopathy syndrome. J of Obstet Anaesth Crit Care. 2014;4(2):50-2.
19. Chhabra A, Jagtap S. Postpartum seizures with posterior reversible encephalopathy syndrome following cesarean delivery for triplets. J Obstet Anaesth Crit Care. 2014;4(1):50-2.
20. Brubaker, Lauren M., et al. ―Hemodynamic and permeability changes in posterior reversible encephalopathy syndrome measured by dynamic susceptibility perfusion-weighted MR imaging.‖ American journal of Neuroradiology 26.4 (2005): 825-830.
21. Karuppannasamy, Divya, et al. ―Cortical visual loss in posterior reversible encephalopathy syndrome in late postpartum eclampsia: Case series.‖ Indian journal of ophthalmology 62.5 (2014): 635.
22. Legriel, S., Pico, F., and Azoulay, E. ―Understanding posterior reversible encephalopathy syndrome.‖ Annual update in intensive care and emergency medicine 2011. Springer Berlin Heidelberg, 2011. 631-653.
23. Sardesai S, Dabade R, Deshmukh S, Patil P, Pawar S, Patil A. Posterior reversible encephalopathy syndrome (PRES): evolvingthe mystery of eclampsia!. The Journal of Obstetrics and Gynecology of India. 2019 Aug;69(4):334-8.
24. Goyal G, Jeswani J. Study of Clinicoradiological Profile in Posterior Reversible Encephalopathy Syndrome: An Experience from North India. Indian Journal of Critical Care Medicine: Peer- reviewed, Official Publication of Indian Society of Critical Care Medicine. 2022;26(4):501.
25. Bembalgi S, Kamate V, Shruthi KR. A study of eclampsia cases associated with

posterior reversible encephalopathy syndrome. Journal of Clinical and Diagnostic Research: JCDR. 2015 Jul;9(7):QC05.

26. Mayama M, Uno K, Tano S, Yoshihara M, Ukai M, Kishigami Y, Ito Y, Oguchi H. Incidence of posterior reversible encephalopathy syndrome in eclamptic and patients with preeclampsia with neurologic symptoms. American journal of obstetrics and gynecology. 2016 Aug 1;215(2):239-e1.
27. Douglas KA, Redman CW. Eclampsia in the United Kingdom. BMJ 1994;308:1395-400.
28. Conde-Agudelo A, Kafury-Goeta AC. Epidemiology of eclampsia in Colombia. Int J Gynaecol Obstet 1998;61:1-8.
29. Katz V, Farmer R, Kuller JA. Preeclampsia into eclampsia: toward a new paradigm. Am J Obstet Gynecol 2000;182:1389-96.
30. Mattar F, Sibai BM. Eclampsia VIII: risk factors for maternal morbidity. Am J Obstet Gynecol 2000;182:307-12.
31. Chames MC, Livingston JC, Ivester TS, Barton JR, Sibai BM. Late postpartum eclampsia: a preventable disease? Am J Obstet Gynecol 2002;186:1174-7.
32. Chen CY, Kwek K, Tan KH, Yeo GS. Our experience with eclampsia in Singapore. Singapore Med J 2003;44:88-93.