WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
Coden USA: WJPRAP Impact Factor 8.63
Volume 15, Issue 11, 791-801. Case Study ISSN 2277–7105

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME IN A POSTPURTAM PRE-ECLAMPSIA PATIENT (PRES): A CASE REPORT

***[1]Chiranjit Das, [1]Batthula Shiva Rani**

Student, Department of Pharmacy Practice, Vaagdevi Pharmacy College, Bollikunta, Warangal, Telangana, India. 506005.

Article Received on 28 April 2026,
Article Revised on 18 May 2026,
Article Published on 01 June 2026,

https://doi.org/10.5281/zenodo.20441675

***Corresponding Author**
Chiranjit Das
Student, Department of Pharmacy Practice, Vaagdevi Pharmacy College, Bollikunta, Warangal, Telangana, India. 506005.

How to cite this Article: *[1]Chiranjit Das, [1]Batthula Shiva Rani. (2026). Posterior Reversible Encephalopathy Syndrome In A Postpurtam Pre-Eclampsia Patient (Pres): A Case Report. World Journal of Pharmaceutical Research, 15(11), 791–801.

ABSTRACT

Background: Posterior Reversible Encephalopathy Syndrome (PRES) is a rare but potentially fatal neurological condition characterized by seizures, headache, altered sensorium, and visual disturbances. It is commonly associated with hypertensive disorders of pregnancy and may occur during the postpartum period due to endothelial dysfunction and impaired cerebral auto regulation. **Case Presentation:** A 26-year-old primiparous woman presented nine days after lower-segment caesarean section performed for fetal distress with two episodes of seizures associated with uncontrolled limb movements, frothing from the mouth, and up rolling of eyes. At admission, she was normotensive; however, she subsequently developed neurological symptoms along with fluctuating blood pressure exceeding 150/100 mmHg. **Investigations:** Magnetic resonance imaging and venography of the brain revealed diffusion restriction in the right posterior parietal lobe and multifocal gyral signal alterations in the bilateral frontal and parietal lobes, suggestive of PRES with associated cytotoxic oedema. Laboratory investigations were within normal limits except for mild electrolyte imbalance. **Management and Outcome:** The patient was managed with magnesium sulphate, anticoagulants, antiepileptics, antihypertensive, and supportive therapy. Following treatment, her clinical condition stabilized without further neurological deterioration. **Conclusion:** This case highlights the significant role of blood pressure fluctuations and endothelial dysfunction in the development of PRES in postpartum pre-eclampsia, even in the

absence of severe hypertension at presentation. Early clinical suspicion, prompt neuroimaging, and timely multidisciplinary management are essential to prevent long-term neurological sequelae and ensure favourable outcomes.

KEYWORDS: Apparent Diffusion Coefficient, Bronchial Artery Embolization, Reversible Cerebral Vasoconstriction Syndrome, Fluid Attenuated Inversion. Posterior Reversible Encephalopathy Syndrome.

1. INTRODUCTION

A rare but dangerous clinical-neuroradiological condition, posterior reversible encephalopathy syndrome (PRES) manifests as a number of nonspecific clinical signs and symptoms, such as headache, vomiting, altered mental status, visual problems, seizures, and unconsciousness. Typically, a seizure manifests as a generalized seizure. Blurred vision, hemianopia, visual neglect, and cortical blindness are among the prevalent visual impairments. These symptoms may develop gradually over a few days or they may be intense. Tendon reflexes are typically active; however, some individuals had diminished or asymmetric limb muscle strength.[1,9]

With a mean age of 45, PRES most commonly affects young or middle-aged adults, while it can occur at any age, from infants to the elderly. Even when patients with eclampsia are excluded, there seems to be a female predominance. PRES can be seen in up to 98% of adult eclampsia patients.[2]

PRES is often associated with reversible cerebral vasoconstriction syndrome (RCVS), which is defined by segmental vasoconstriction and vasodilation in small cerebral arteries due to dysregulation of cerebral vascular tone. The finding that hypertensive crises can result in PRES raises the possibility that the abrupt increase in blood pressure seen in RCVS plays a crucial role in the development of PRES. Another factor contributing to the higher incidence of PRES in patients undergoing immunosuppressive therapies, sepsis, preeclampsia/eclampsia, and cytotoxic drug therapy is endothelial dysfunction, which can be caused by endogenous or exogenous toxins.[3,10] Pregnancy and puerperal illnesses, organ transplantation, immunosuppressive or cytotoxic drugs, acute or chronic renal disease, autoimmune diseases, infections, endocrine disorders, etc. are among the risk factors for developing PRES.[4,11]

The actual mechanism of PRES is still mostly unknown. There are two conflicting pathophysiological mechanisms described. The vasospasm/hypoperfusion theory comes first. This hypothesis states that the various risk factors produce vasoconstriction or vasospasm, which in turn causes brain hypoperfusion and cerebral ischemia, which in turn creates greater brain vasogenic oedema. The hypertension/hyperperfusion hypothesis, on the other hand, is the second theory. This theory states that severe hypertension that beyond the limit of vascular auto regulation causes vasogenic oedema in brain tissue, which leads to excessive perfusion, vascular endothelial damage, and small artery passive expansion.[12,13]

The placenta plays a crucial part in the pathogenesis of pre-eclampsia, even if the exact pathophysiology is unknown. Tumour necrosis factor (TNF-α), interleukin (IL)-1, interferon (IFN)-γ, and IL-6 are among the inflammatory cytokines that are released into the mother's blood circulation by activated T-helper cells and placental ischemia, which causes more syncytial surface tissue apoptosis, necrosis, and dropping off as the placenta forms. This causes a severe maternal systemic immune response (toxaemia of pregnancy), which in turn causes systemic endothelial cell activation and damage. Large volumes of vasoconstrictor and inflammatory mediators are secreted by activated endothelial cells, which also generate widespread systemic vascular contraction and cerebral vasogenic oedema.[5,14]

Neuroimaging is used in conjunction with clinical evaluation to diagnose PRES. When using T2-weighted and Fluid-Attenuated Inversion Recovery (FLAIR) sequences, magnetic resonance imaging (MRI), the gold standard, shows bilateral, symmetrical hyperintensities, primarily in the parietal and occipital lobes. Although cytotoxic oedema may manifest in 10–15% of patients, diffusion-weighted imaging (DWI) usually indicates no limitation, differentiating PRES from ischemic stroke. Although it lacks the sensitivity of MRI, computed tomography (CT) can identify hypodensities in emergency situations.[6,15]

Because PRES is uncommon and develops quickly, there are presently no randomized controlled studies for its management or treatment. The goal of treatment is to find and treat the underlying reason. This includes controlling blood pressure, employing antiepileptics, modifying prescriptions, fixing electrolyte imbalances, and making sure pregnant women receive timely care. To prevent ischemia in hypertensive conditions, blood pressure must be gradually lowered. Magnesium sulphate is a good choice for pregnant women with preeclampsia or eclampsia; antiseizure drugs should be chosen depending on the patient's renal function and any adverse effects.[7,16]

2. CASE PRESENTATION

Subjective findings

A 26 year of female patient presented to the hospital on 13th September 2025 with chief complaint of 2 episodes of seizure activity after post Lower segment caesarean section (04/09/25). She has experienced 1st seizure activity on for 2-3 minutes and she got 2nd episode early morning for 1-2 min, focal seizure was positive, apart from those she has presented with involuntary limb movements (left foot), froth from mouth and up rolling of eyes positive.

Obstetric history

A healthy primi male baby was born on 04/09/25 with the body weight 2.5 kg. Indication for LSCS- Cord around the neck with fetal distress.

Initial management

Initially to reduce complaints of the patients (inj. levera it is given at a dose of 1 gm twice a day Intravenously, inj. Pantop is administered at a dose of 40 mg once a day IV, inj. Mannitol is given at a dose of 100 ml four times a day IV and Inj. lacosamide given at a dose of 200 mg twice a day IV) are prescribed.

Table 1: Day wise vitals & systemic examination details of the patient.

VITALS	DAY1	DAY 2	DAY 3	DAY 4	DAY 5	DAY 6	DAY 7
ON EXAMINATION/ COMPLAINTS	Sedation	C/C	Neck pain	C/C	Pt. is drowsy, arousable	Pt. is irritable	c/c
TEMPERATURE	Afebrile	98°F	98°F	Afebrile	Afebrile	Afebrile	Afebrile
BLOOD PRESSURE (mmHg)	110/70	150/100	150/100	130/80	130/90	110/70	120/80
PULSE RATE (bpm)	95	78	74	94	95	74	80
RESPIRATORY SYSTEM	BAE +	BAE +	BAE +	BAE +	BAE +	BAE +	BAE +
CARDIOVASCULAR SYSTEM	S1 & S2 +	S1 & S2 +	S1 & S2 +	S1 & S2 +	S1 & S2 +	S1 & S2 +	S1 & S2 +
PER ABDOMEN	Tender	Soft	Soft	Soft	Soft	Soft	Soft

Laboratory investigations

Table 2: Haemogram.

HAEMOGLOBIN	13.9 gms%	(12 - 18)
MCV	81.8 fL	(76 - 96)
MCH	26.1 pg	(27 - 32)

Serum Electrolytes

- ✓ Serum sodium (Na^+)- 134 mmol/L
- ✓ Serum potassium (K^+)- 3.4 mmol/L
- ✓ Serum magnesium (Mg^{++})- 1.5 mg/dL

ECG

- ➢ Sinus tachycardia
- ➢ Ventricular premature complexes
- ➢ Consider right atrial enlargement
- ➢ Probable left ventricular hypertrophy.
- ➢ Borderline prolonged QT interval.
- ➢ Abnormal ECG

MRI Brain with MR Venogram

Impression

- Focal gyriform area of diffusion restriction with low ADC values involving the right posterior parietal region – likely represent cytotoxic oedema /Infarct.
- Multifocal areas of gyral signal changes involving the bilateral frontal, parietal regions, bilateral caudate nucleus and bilateral posterior parietal lobes.
- Normal MR Brain venogram.
- Diagnostic possibilities include:

- ➢ PRES (Posterior Reversible Encephalopathy Syndrome).
- ➢ Associated Anticardiolipin Antibody Syndrome.
- ➢ As compared to previous MRI study dated: 08/09/2025, there is reduction of the signal changes with residual signal changes noted in the present study.

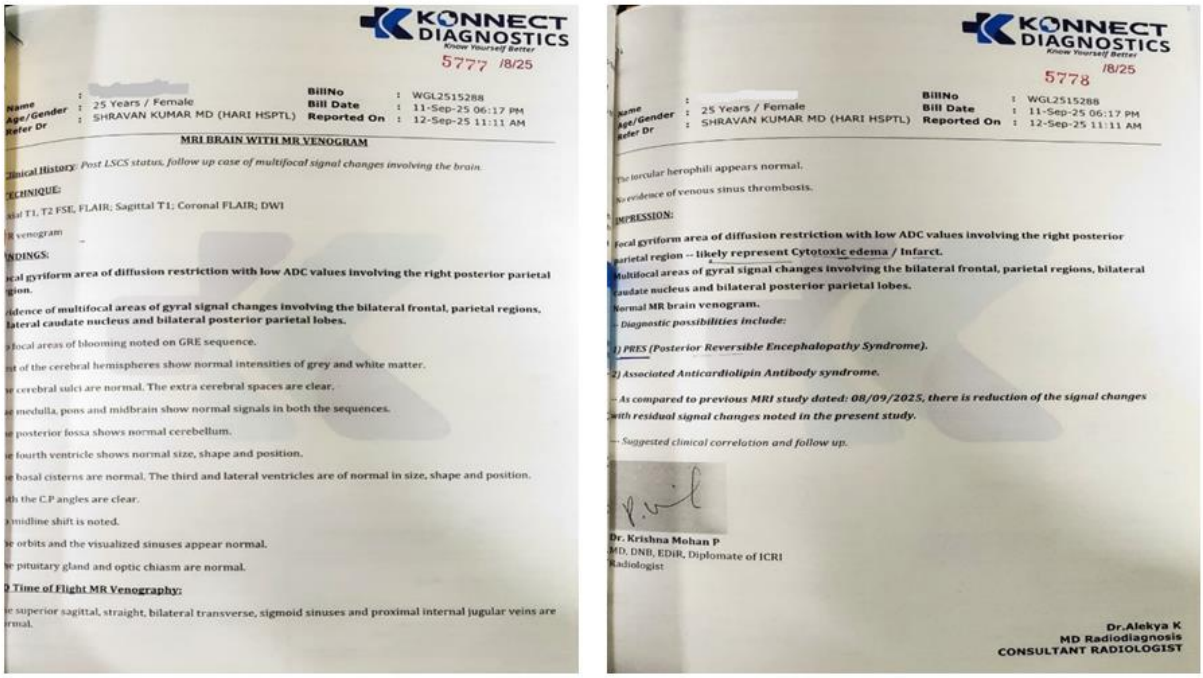

Figure 1: Evidence of the MRI brain with MR Venogram.

Table 3: Treatment chart.

S.No.	BRAND NAME	GENERIC NAME	DOSE	ROA	FREQ
1.	INJ. LEVERA	Levetiracetam	1gm	IV	BD
2.	INJ PANTOP	Pantoprazole	40 mg	IV	OD
3.	INJ MANNITOL	Osmotic mannitol	100ml	IV	QID
4.	TAB.ECOSPIRIN	Aspirin	75 mg	PO	OD
5.	INJ MgSo4	Magnesium sulphate	1gm	IV	BD
6.	INJ. CLEAXANE	Enoxaparin	40 mg	IV	OD
7.	INJ.DEXA	Dexamethasone	8 mg	IV	BD
8.	INJ. MONOCEF	Ceftriaxone	2 gm	IV	BD
9.	INJ. VIMPAT	Lacosamide	200mg	IV	BD
10.	INJ. ENCHORATE FORTE	Valproate	7500mg	IV	BD
11.	INJ. MAGNEX FORTE	Cefoperazone +Sulbactam	1.5 mg	IV	BD
12.	INJ. LORAZEPAM	Lorazepam	1mg	IV	STAT
13.	TAB. NORMODYNE	Labetalol	100mg	PO	OD
14.	TAB. PERAMPA	Perampanel	4 mg	PO	HS
15.	TAB. APRESOL	Dihydralazine	25 mg	PO	OD
16.	TAB. CLONOTRIL	Clonazepam	0.25 mg	PO	OD
17.	TAB. NEXITO	Escitalopram	5 mg	PO	BD
18.	TAB. ZERODOL TH	Aceclofenac thiocolchicoside	1tab	PO	BD
19.	TAB ECOSPIRIN AV	Aspirin +Atorvastatin	150/20 mg	PO	OD
20.	FLEXABENZ GEL	Cyclobenzaprine hydrochloride	-	LA	TID
21.	TAB. NICARDIA	Nifedipine	10 mg	PO	TID
22.	SYP. KCL	Potassium Chloride	15 mg	PO	TID

23.	TAB. EVION- LC	Levocarnitine and Vitamin E	1 Tab	PO	BD
24.	TAB. MONOCEF-O	Cefpodoxime proxetil	200 mg	PO	BD
25.	TAB. QUETAPINE	Quetapine fumarate	25 mg	PO	BD

Course in the hospital

Patient went to the outside hospital. MRI brain with venogram done showing features suggestive of PRES syndrome and referred here for further management. CT brain done on admission showing no significant abnormally. 2D Echo showing normal study. Blood investigation revealed normal study. Serum, Ammonia level WNL. Neurophysician opinion taken and on his advice samples sent for S. ANA profile, homocysteine levels and ANC A antibodies levels, reports awaited. Psychiatrist opinion taken and his orders followed. During course in hospital stay, patient treated with Antiepileptics, Anti-HTN, Antibiotics, Inj. monocef, Anticoagulants and other supportive medication. Patient became hemodynamically better but needs further hospitalization in view of persistent irritability but attenders are not willing and hence being discharge at request.

Table 4: Discharge medication.

S.NO	DRUG NAME	DOSE	FREQ	R.O. A	DURATION
1.	Tab. Monocef	200 mg	BD	PO	5 Days
2.	Tab. Levipil	75 mg	BD	PO	5 Days
3.	Tab. Ecospirin-AV	150/20 mg	OD	PO	5 Days
4.	Tab. Encorate	500 mg	BD	PO	5 Days
5.	Tab. Lacosamide	100 mg	BD	PO	5 Days
6.	Tab. Pantocid	40 mg	OD	PO	5 Days
7.	Tab. Perampa	2 mg	OD	PO	5 Days
8.	Tab. Apresol	25 mg	OD	PO	5 Days
9.	Tab. Nicardia	10 mg	TID	PO	10 Days
10.	Syp. Potclor	15 ml in 1 glass of H_2o	TID	PO	3 Days
11.	Tab. Clonotril	0.25 mg(1/2-1/2-1 tab)	TID	PO	5 Days
12.	Tab. Evion-LC	1 tab	BD	PO	5 Days
13.	Tab. Nexito	5 mg	BD	PO	5 Days

3. DISCUSSION

The severity of HTN in PRES patients varies and is rarely excessive. Furthermore, blood pressure variations (blood pressure fluctuation) prior to PRES onset is no more likely than in other individuals with a history of HTN.[8,18] Two-thirds of women with uncomplicated pregnancies get PRES, a rare disease, in the first week following delivery. Additionally, individuals with severe pre-eclampsia may occasionally experience it. Endothelial damage as well as impaired cerebro-vascular auto regulation is pathophysiologically linked to vasogenic oedema in the postpartum pre-eclampsia phase in our patient, especially in the posterior area,

which is more susceptible to changes in systemic blood pressure fluctuation. As seen in this case, localized ischemia damage or seizures may cause this process to advance to secondary cytotoxic oedema.[9]

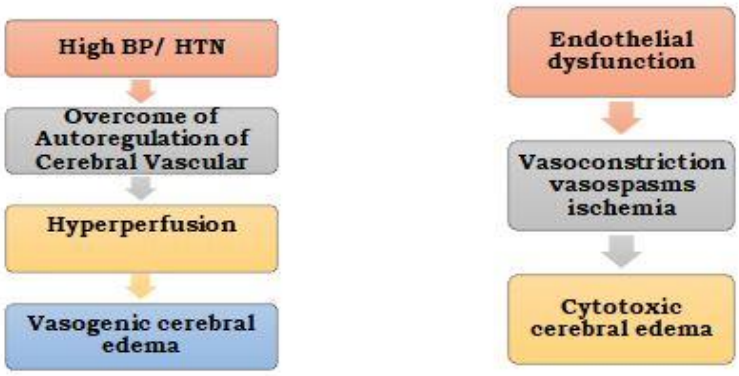

Fig. 2: A figure that summarizes the PRES processes. (Two theories).

The multiorgan condition known as pre-eclampsia is linked to morbidity and death in both the mother and the fetus. Pre-eclampsia is indicated by neurological symptoms that appear during the postpartum period. PRES should always be considered when a patient in the postpartum phase exhibits a combination of seizures, blurred vision, and headache.[20]

On admission, our patient's blood pressure was normal; nevertheless, the next day, it steadily climbed to 150/100, And before nine days, this patient gave birth to a child through LSCS. The patient had blood pressure fluctuations and neurological symptoms (two episodes of seizures with involuntary limb movement's froth from her lips, and up rolling of her eyes and experiencing focal seizures with movement of her left limb) throughout the post-LSCS period, which was indicative of postpartum pre-eclampsia exacerbated by PRES. We postulated that one of the main risk factors for the development of PRES is high blood pressure, together with blood pressure fluctuations.

The brain MRI with venogram shows low ADC values in the right posterior parietal region and a localized gyriform area of diffusion restriction, which are probably signs of cytotoxic oedema or infarct. Further evidence of PRES syndrome includes multifocal gyral signal alterations in the bilateral frontal and parietal areas, as well as the bilateral caudate nucleus and posterior parietal lobes. Serum ammonia levels were within normal ranges, additionally, the electrolytes (sodium, potassium, and magnesium) in the serum are steadily declining. A

normal 2D echocardiography and blood tests were recorded, and a CT scan performed upon admission revealed no notable abnormalities.

The patient was treated with antiepileptics during her hospital stay for repeated focal and generalized seizures linked to PRES, which progressed to status epilepticus. Among other supportive medications, antihypertensive therapy was started to control postpartum blood pressure fluctuations and prevent cerebral hyperperfusion; antibiotics were given because of the patient's post-LSCS status to prevent the risk of post-operative infection; anticoagulants were given as a thromboprophylaxis to prevent thromboembolic complications in this postpartum post-LSCS patient; and injections of mannitol and dexamethasone were given to control vasogenic cerebral oedema or cytotoxic oedema and to eliminate the risk of intracranial pressure (ICP). This patient was treated with an intravenous injection of magnesium sulfate and an oral potassium supplement to prevent seizures and treat hypomagnesemia and hypokalaemia.

4. CONCLUSION

This particular case developed PRES due to blood pressure fluctuation after the post LSCS of the patient which is an unusual circumstance compared to the patient's HTN history. This example demonstrates that PRES can develop even in cases when blood pressure is not appreciably elevated at presentation, with endothelial dysfunction and blood pressure fluctuations playing a significant role. This instance emphasizes how postpartum patients who have seizures or other neurological symptoms require timely MRI imaging and early clinical suspicion. Favourable outcomes and the prevention of permanent neurological sequelae can be achieved through timely management of blood pressure control, antiepileptic therapy, and supportive care.

5. CONSENT

All authors declare that written informed consent was obtained from the patient for this case report and images.

6. ETHICAL APPROVAL

It’s not applicable.

7. ACKNOWLEDGEMENT

The authors sincerely thank the patient for their cooperation, trust, and consent to share clinical details for academic and educational purposes. Their willingness to contribute to this report is gratefully acknowledged.

8. COMPETING INTERESTS

Authors have declared that no competing interests exist.

9. REFERENCES

1. Zhang, L., Wang, Y., Shi, L., Cao, J., Li, Z., & Wáng, Y. X. (2015). Late postpartum eclampsia complicated with posterior reversible encephalopathy syndrome: a case report and a literature review. *Quantitative imaging in medicine and surgery*, *5*(6): 909–916.
2. Practical Neurology Triplett, J. D., Kutlubaev, M. A., Kermode, A. G., & Hardy, T. (2022). Posterior reversible encephalopathy syndrome (PRES): Diagnosis and management. *Practical Neurology, 22*(3): 183–189.
3. Internal Medicine Ando, Y., Ono, Y., Sano, A., Fujita, N., & Ono, S. (2022). Posterior reversible encephalopathy syndrome: A review of the literature. *Internal Medicine*, *61*(2): 135–141.
4. Quantitative Imaging in Medicine and Surgery Chen, Z., Zhang, G., Lerner, A., Wang, A. H., Gao, B., & Liu, J. (2018). Risk factors for poor outcome in posterior reversible encephalopathy syndrome: Systematic review and meta-analysis. *Quantitative Imaging in Medicine and Surgery, 8*(4): 421–432.
5. Barbhuiya, S. M., & Ali, S. K. A. (2025). Posterior reversible encephalopathy syndrome in obstetrics: Pathophysiology, diagnosis, and management considerations. *International Journal of Clinical Obstetrics and Gynaecology, 9*(2): 100–103.
6. Journal of Stroke and Cerebrovascular Diseases Rabinstein, A. A., Mahendrakar, J., Merrell, R., Kozak, O. S., Durosaro, O., & Fugate, J. E. (2011). Blood pressure fluctuations in posterior reversible encephalopathy syndrome. *Journal of Stroke and Cerebrovascular Diseases*, *21*(4): 254–258.
7. Ayadine, A., Oukili, H., Benali, S., Messaoudi, H., Elhassani, M., & Kouach, J. (2024). Post-partum eclampsia complicated with a posterior reversible encephalopathy syndrome: A case report. *EC Gynaecology, 13*(12): 1–5.

8. BMJ Case Reports Verhaegen, J., Peeters, F., Debois, P., & Jacquemyn, Y. (2019). Posterior reversible encephalopathy syndrome as a complication of pre-eclampsia in the early postpartum period. *BMJ Case Reports*, *12*(7): 228954.
9. Pradeep, S., & Mitta, S. G. (2024). An Overview on Scleroderma. Glob Acad J Med Sci., 6(6): 320-326.
10. Mitta, S. G., Tanvi, S., Kavya, A., Rahaman, S., & Alam, K. (2025). Recurrent Squamous Cell Carcinoma of the Scalp: A Case Report Emphasizing Early Detection. Journal of Drug Delivery & Therapeutics, 15(12).
11. Rohini, S. Chiranjit Das, & Sandeep Goud Mitta. (2024). A case report on arthritis induced inflammatory bowel disease. Glob Acad J Med Sci., 6.
12. Mitta, S. G., Elagonda, S., Koluguri, A. S., & Nagunuri, H. (2025). Pharmacovigilance in Action: Antibiotic-Induced Stevens–Johnson Syndrome in Primary Care. Asian Journal of Research in Dermatological Science, 8(1): 192-197.
13. Mitta, S. G., Tanvi, S., Bhargavi, U., & Ruchitha, V. (2024). An Overview on Pulmonary Insulin. Glob Acad J Pharm Drug Res., 6: 12-19.
14. Charan, M. S., Swathi, D., Hyndavi, A., & Mitta, S. G. (2025). Hallux Valgus Bursitis: Clinical Presentation and Management of a Rare Case. International Journal of Medical and Pharmaceutical Case Reports, 18(4): 83-87.
15. Charan, M. S., & Mitta, S. G. (2025). Advances in the Understanding of Protein C and Protein S Deficiencies: from Molecular Mechanisms to Therapeutic Strategies. International Journal of Research and Reports in Hematology, 8(2): 383-393.
16. Das, C., Aktar, N., Rohini, S., & Mitta, S. G. (2025). Proton Pump Inhibitors and Gut Microbiota: A Comprehensive Review of Microbial Alterations and Clinical Implications. Asian Journal of Research and Reports in Gastroenterology, 8(1): 318-326.
17. Swetha, M., Sahithi, K., Lahari, M., Yashwanth, P., Nabila, M., & Mitta, S. G. (2024). DEVELOPMENT AND VALIDATION OF UV VISIBLE SPECTROPHOTOMETRIC METHOD FOR DETERMINATION OF LAMOTRIGINE IN BULK AND PHARMACEUTICAL DOSAGE FORMS.
18. Hamsarekha, P., Anuhya, B., Bandhavi, K., Snehanjali, M., Thatipelli, R. C., & Mitta, S. G. (2023). Viral Hepatitis–A Review Article. *International Journal*, *6*(5): 111.
19. Hamsarekha, P., Anuhya, B., Bandhavi, K., Snehanjali, M., Thatipelli, R. C., & Mitta, S. G. (2023). Viral Hepatitis–A Review Article. *International Journal*, *6*(5): 111.
20. Mitta, S. G., Chandana, B., Snehanjali, M., Paidi, M., Karim, A., Sukanya, A., & Shylaja, K. (2025). A SHORT REVIEW ON CLINICAL DATA MANAGEMENT.