

Case Report

Lightening Strikes Twice: A Spectrum of Posterior Reversible Encephalopathy Syndrome and Reversible Cerebral Vasoconstriction Syndrome

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Abstract: Background: Posterior reversible encephalopathy syndrome (PRES) and reversible cerebral vasoconstriction syndrome (RCVS) although two independent clinical entities but can present in the same patient as a continuum of the same disease process. PRES can be characterized by varied neurological symptoms such as headache, impaired visual acuity or visual field deficit, confusion, impaired consciousness, seizures, and motor deficits. Ischemic infarctions in the territory of vasogenic edema post PRES is not uncommon. RCVS is typically associated with severe thunderclap headaches and reversible segmental vasoconstriction of cerebral arteries and is often complicated by ischemic or hemorrhagic stroke. More research is required to clearly delineate the link between them. **Case:** Here we report the case of a normotensive lady who in her post partum period developed severe holocranial headache with seizure and confusional state. A MRI Brain done showed T2 /FLAIR hyperintensities in bilateral parieto-occipital and frontal lobes suggestive of vasogenic edema and a diagnosis of PRES was made in her initial admission and she improved with conservative management. But soon after she again developed headache followed by dysarthria, left more than right sided weakness and right visual inattention and right-left disorientation. MRI Brain done now showed changes suggestive of PRES with associated infarcts. But a CT angiography done now showed evidence of intracranial vasospasm. Patient was diagnosed and managed like RCVS. However, no underlying cause of PRES/RCVS could be determined in her case except her postpartum period which could be considered as risk factor for both these conditions. **Conclusion:** This case highlights that PRES and RCVS although relatively uncommon neurological disorders, they share some common clinical and radiologic features and dilemma arises when clinico-radiological features suggestive of both conditions develop in same patient like seen in our case. Therefore, the two conditions can be thought of as a manifestation of same underlying disease process and further studies are required to establish this hypothesis. **Keywords:** Headache, PRES, RCVS, pregnancy, seizure, T2 FLAIR Hyperintensity, Vasogenic Edema, Intracranial Vasospasm.

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BACKGROUND

Reversible cerebral vasoconstriction syndrome (RCVS) and posterior reversible encephalopathy syndrome (PRES) are being increasingly recognised neurological disorders owing to widespread use of brain magnetic resonance imaging (MRI) and more clinical awareness.

Although the pathophysiology of PRES remains controversial, the mechanism of a rapid increase in blood pressure is supposed to be central causing disruption of blood flow autoregulation and cerebral vasogenic edema. Therefore, PRES is often seen to occur

on a background of hypertension or rising blood pressure, although normotensive or hypotensive patients with PRES have also been reported (Pilato *et al.*, 2020). Risk factors of PRES include immunosuppression, malignancy, pre-eclampsia, renal failure, autoimmune disorders, sepsis, hypertension, transplantation, and chemotherapeutic medications and may also occur in healthy subjects (Pilato *et al.*, 2020). PRES may develop at any age from infants to the elderly, but it usually affects young or middle-aged adults, with a female preponderance (Lee *et al.*, 2008). PRES patients may present with several neurological symptoms like headache, impaired visual acuity, or visual field deficits,

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confusion, focal neurological deficits, and disorders of consciousness and seizures. (Pilato *et al.*, 2020). Typical CT and MRI Brain in PRES would show symmetrical hemispheric vasogenic oedema affecting subcortical white matter and often extending to the overlying cortex, best seen on MRI fluid-attenuation inversion recovery (FLAIR) sequences (Casey SO *et al.*, 2000) and confirmed by MRI diffusion-weighted imaging with absence of restricted diffusion. Postcontrast enhancement occurs in 38%–50% of patient (Bartynski & Boardman, 2007). Usually, vasogenic edema is the hallmark of PRES even if small areas of cytotoxic edema may occur in neuroimaging. (Al-Sherif, 2015). Although reversibility of radiological signs is a hallmark feature of PRES, sometimes the vasogenic edema progresses to permanent tissue damage causing cerebral infarction.

Reversible Cerebral Vasoconstriction Syndrome (RCVS), previously known as isolated benign cerebral vasculitis, or Call-Fleming syndrome are a group of syndromes characterized by severe headaches, typically associated with reversible segmental constriction of cerebral arteries, which may become complicated by ischemic or hemorrhagic stroke (Pilato *et al.*, 2020). RCVS can be diagnosed based on key clinical features of thunderclap headache (or severe recurrent headache), cerebral vasoconstriction demonstrable on imaging in at least 2 different cerebral arteries and resolution of this vasoconstriction by 3 months, in the absence of primary angiitis of the central nervous system (PACNS), or aneurysmal subarachnoid hemorrhage (SAH) (Burton & Bushnell, 2019). The pathophysiology of RCVS has not been clearly elucidated but a possible role of a transitory cerebral vascular autoregulation dysfunction and blood–brain barrier (BBB) breakdown have been postulated. Conditions associated with RCVS are -pregnancy, eclampsia, neurosurgical procedure, and vasoactive drug use. RCVS commonly involves women between the ages of 20 and 50 and is usually self-limiting but recurrences and complications may occur (Pilato *et al.*, 2020).

Bilateral symmetric parieto-occipital lesions seen on MRI is typical for PRES, but not characteristic for the RCVS. The classical radiological presentation assessed by MRA or conventional angiography includes cerebral vasoconstriction, with at least two narrowings in the same artery, on two different cerebral arteries; commonly, these arterial abnormalities disappear in 3 months (Ducros *et al.*, 2007). Cerebral catheter digital subtraction angiography (DSA) is considered the gold standard for visualizing vasoconstriction. SAH or intraparenchymal hemorrhage are common complications of RCVS (Pilato *et al.*, 2020). In RCVS, symptoms typically follow a self-limiting course, with resolution by 3 weeks but resolution of vasoconstriction may take 3 months.

RCVS and PRES have been previously described in the literature as two associated entities.

They share similar triggers, clinical and radiological features and may have similar pathophysiological mechanisms (Jeanneret *et al.*, 2022). Whether PRES and RCVS are two independent overlapping entities or they form a continuum is still debated. The pathophysiological mechanisms in both conditions have been associated with blood flow dysregulation, endothelial dysfunction, which leads to breakdown of the BBB, interstitial fluid extravasation, and vasogenic edema. The dysregulation of cerebral arterial tone can also lead to segmental vasoconstriction, hypoperfusion, infarction, and cytotoxic edema (Jeanneret *et al.*, 2022). Autopsy studies in patients with PRES have shown intimal thickening, segmental vessel narrowing, and thrombi formation (Ducros *et al.*, 2007), thus, supporting the idea that these two conditions represent a continuum of the same disease process. In another study done by Purohit *et al.*, they found arterial stenosis on imaging in 28% (13/46) of PRES cohort (15) drawing similarities with reversible vascular narrowing found in RCVS. Here our patient presented with overlapping features of these two conditions indicating common underlying pathophysiological mechanisms of these two entities and supporting the presence of a PRES-RCVS spectrum. The only underlying common risk factor for PRES/RCVS in our patient was pregnancy/postpartum. What was unusual in our patient was the absence of thunderclap headache (TCH) in presentation which is a classical hallmark of typical RCVS.

CASE PRESENTATION

We present the case of a 28 year old lady P2+0 with no known comorbidities who delivered her second born on 16.6.25 by LUCS and subsequently on morning of 17.6.25 she developed a severe holocranial headache with nausea and vomiting and had a generalized tonic clonic seizure with post ictal confusion. She was admitted here at our hospital 2 days later. Her BP was in the range 120-140 mm hg systolic and 80-90 mm hg diastolic and other vitals were recorded as in normal range. On neurological examination she was found to have a mild confusion but no other focal neurological deficit. A CT Brain was done on admission which did not show any apparent abnormality. Her pregnancy had been uneventful with no history of hypertension/pre-eclampsia/eclampsia, fever with rash, or any other acute illness. She was managed emergently with antiseizure medications and magnesium sulphate. A MR Venogram was done which showed- Cortical altered signal intensity in the form of T2 FLAIR hyperintensity and T2 shine symmetrically involving bilateral parieto-occipital and posterior frontal lobes without obvious post contrast enhancement and an otherwise normal MR venogram (**FIGURE 1: DWI and FLAIR hyperintensity in bilateral parieto-occipital lobes**) (**FIGURE 2: Normal MR Venogram**). These changes showing presence of vasogenic edema predominantly in the posterior cortex were suggestive of PRES (Posterior Reversible Encephalopathy Syndrome)/Post ictal changes. Her EEG was suggestive of a diffuse cortical dysfunction. A CSF

study was done to evaluate for any infectious / inflammatory etiology. CSF showed 3 cells, 100% lymphocytes, protein- 79mg/dl, glucose -56 mg/dl, ADA 0.8. As there was no evidence of any infective changes in the CSF and a elevated CSF protein can be reflective of the breakdown of the blood brain barrier in Posterior Reversible Encephalopathy Syndrome (PRES)- the patient was diagnosed and managed as PRES. A extensive evaluation for the etiology of PRES was undertaken which showed- ANA 4+ in 1:100 dilution, in homogenous pattern, but ANA panel was negative for any specific antibody. Results of ANCA, RA factor, Anti CCP antibodies, Lupus anticoagulant, anti-beta 2 glycoprotein IgG and IgM, anti-Cardiolipin antibody IgG and IgM were all nonreactive, but serum C3 and C4 levels were elevated. She had no history of any polyarthralgia, rash, oral or genital ulcers. Although the presence of ANA (Antinuclear Antibodies) are linked with connective tissue diseases, often such ANA positivity maybe detected in healthy individuals (Grygiel-Górniak *et al.*, 2018). Such ANA positivity may indicate a underlying overactive immune system and not necessarily indicate any autoimmune disorder. According to literature, the percentage of ANA positivity in the general population can be approximately 25% by using indirect immunofluorescence microscopy

performed on HEp-2 cells (IIFA on HEp-2 or HEp-2000) (WANDSTRAT *et al.*, 2006). According to the American College of Rheumatology (ACR) and the European League against Rheumatism (EULAR) cut off criteria for SLE is positive ANA at a titer of 1: 80 or greater (Pisetsky & Lipsky, 2019). Although a relatively high level of significance is set for ANA titre values to be considered relevant in diagnosing connective tissue diseases, the incidence of a significantly elevated ANA level in the general population is still found to be 2.5% (WANDSTRAT *et al.*, 2006). Therefore, a positive ANA value even in high titre needs to be interpreted with caution as most people with a positive ANA count are not diagnosed with autoimmune diseases, and the probability of future disease is also low (Grygiel-Górniak *et al.*, 2018). While ANA positivity have been reported in PRES patients with underlying SLE and lupus nephritis (DZIRI *et al.*, 2022) (Wang *et al.*, 2023), there have been no direct causative links established between PRES and ANA positivity. So, in our patient the ANA positive status was deemed to be incidental in the absence of any other evidence of underlying connective tissue diseases, and no specific therapy for it was instituted. She symptomatically improved with conservative management of her PRES and she was subsequently discharged after 5 days.

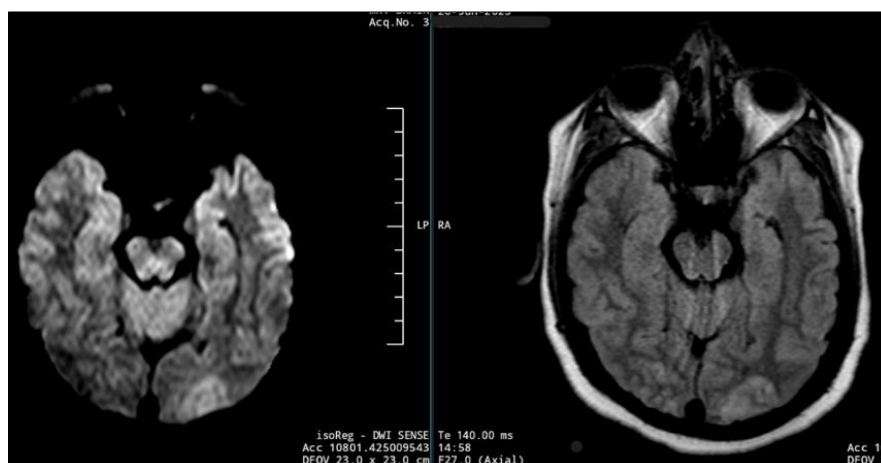


Figure 1

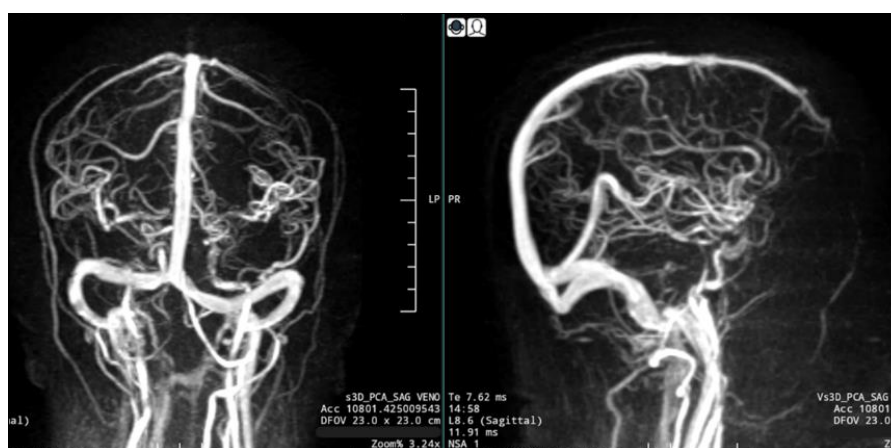


Figure 2

Now since early morning 2AM of 28.6.25 our patient again developed a headache followed by a generalized weakness and gait instability such that she could not get up from bed and mobilize on her own. She also developed some difficulty visualizing objects on the right side of her visual field. She could not state any facial deviation but had some slurring of speech and her weakness was more on left side. When she was brought to our ER on evening of 28.6.25- she was found to have a GCS-15/15, left sided facial UMN palsy, mild dysarthria, left sided upper and lower limb weakness was more pronounced (power-2/5) than the right side (power 4/5), left plantar response was extensor, and there was visual inattention to right sided and some right- left disorientation.

An urgent MRI Brain was done which revealed asymmetrical T2/FLAIR cortical as well as subcortical hyperintensities involving bilateral frontal, parietal, occipital lobes sparing deep grey matter, brainstem, and cerebellum. There was also asymmetric diffusion restriction in bilateral parietooccipital cortex and deep watershed region, with no abnormal enhancement post contrast (**FIGURE 3A, 3B**: Diffusion Restriction in DWI in bilateral parietooccipital cortex). These MRI features were suggestive of PRES with associated infarcts, and compared to the previous study 7 days back, now the area involved had increased with new onset infarcts. The areas of vasogenic edema in her previous MRI Study done a week back had evolved now to

cytotoxic edema and infarctions. A subsequent CT Angiography of cerebral vessels was done which showed- diffuse caliber narrowing of all the intracranial vessels of both anterior and posterior circulation indicating diffuse intracranial vasospasm (**FIGURE 4**: Diffuse narrowing of intracranial vessels of both anterior and posterior circulation seen indicating diffuse intracranial vasospasm). However Doppler study of both Carotid and Vertebral arteries did not reveal any significant stenosis. Her EEG study showed encephalopathic pattern with no epileptiform abnormalities. PRES leading to subsequent infarcts was our initial diagnosis in her case this time. But her CT Angiography study showing the diffuse vasospasm of cerebral vessels was suggestive of a diagnosis of RCVS(Reversible Cerebral Vasoconstriction Syndrome) and in the absence of any other definite underlying cause, postpartum angiopathy was thought to be the possible etiology of the RCVS in her case. Although this time the patient did not have any significant headache and presented with neurodeficits correlating with her infarctions, she was treated with Nimodipine for her suspected RCVS. The patient improved symptomatically with conservative management with Nimodipine and antiseizure medications. Her hemianopia and visual inattention resolved and the power in her limbs improved to 5/5 on both upper and lower limbs bilaterally. She was subsequently discharged with advice to follow up with us in OPD.

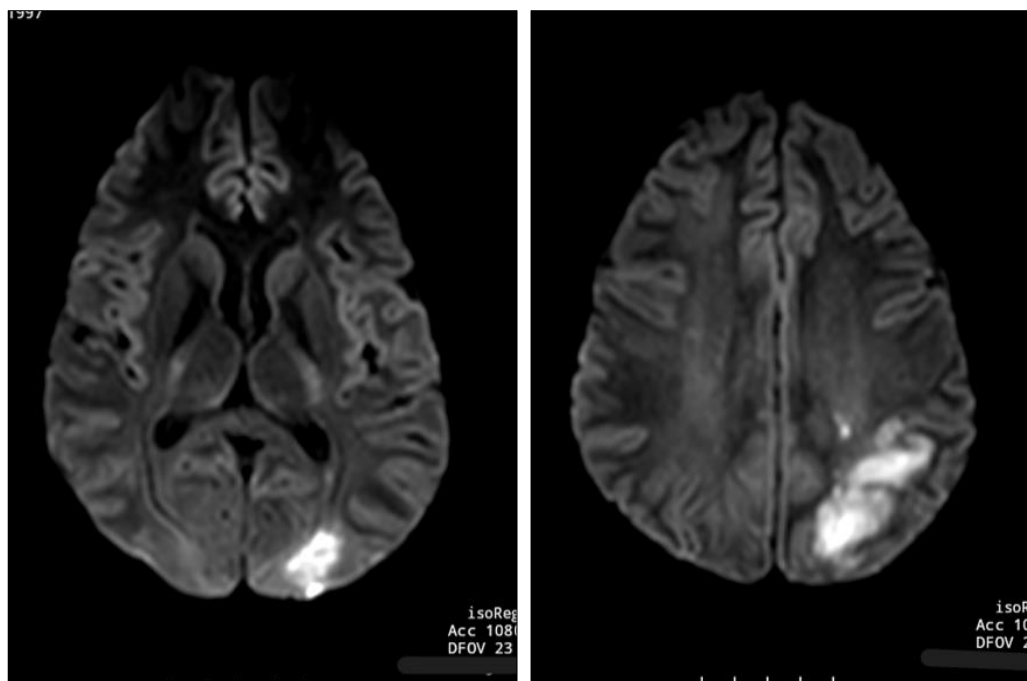


Figure 3a and 3b

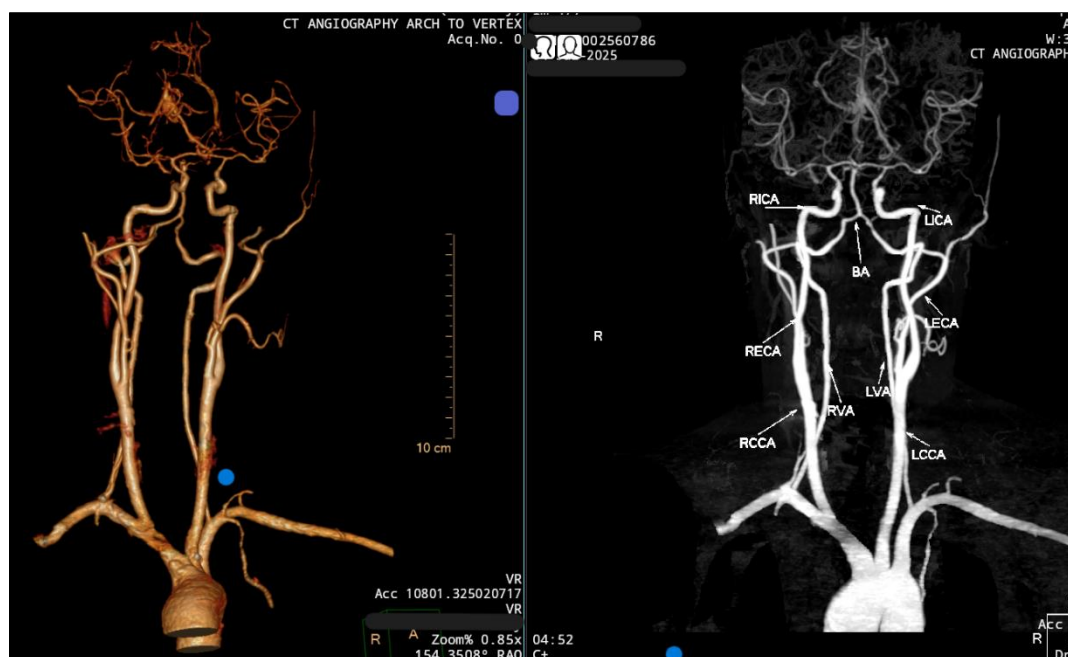


Figure 4

What was unusual in our patient was the absence of thunderclap headache (TCH) in presentation which is a classical hallmark of typical RCVS. Literature states that TCH is usually the initial symptom of RCVS and that up to 82% to 100% of patients with RCVS have repeated attacks of thunderclap headaches (Choi *et al.*, 2018). However, atypical presentation of RCVS has been reported where TCH have been absent like a case reported by Choi E *et al.*, (Choi *et al.*, 2018). An Italian study showed that upto 30% of the RCVS patients in the study did not have TCH and those without TCH were associated with poorer cerebrovascular complications as compared to those with TCH (Lange *et al.*, 2022).

What was also atypical in our patient was a recurrence of symptoms within days of resolution of the first episode in the absence of any identified risk factor. Recurrent PRES is very rare in the literature, and end stage renal disease has been found to be the underlying risk factor in a few cases (Komur *et al.*, 2012). To prevent relapse of PRES it is advised to carefully monitor and control the BP which was done in our patient and to identify and treat any underlying cause. When we looked retrospectively in our case, as in what intervention could have been done from our side to prevent the relapse in our patient, it seemed to us that looking for vasospasm in the background of PRES by CT angiography or DSA could have been done in her first admission as according to reports 87% of PRES cases can have angiographic changes consistent with RCVS, such as diffuse vasoconstriction, focal vasculopathy or vessel pruning (Bartynski, 2008). Therefore, if such vasospasm had been detected in our patient during her first admission, therapy for it could have been instituted, which may have prevented the relapse.

In a study published by Singh *et al.*, the risk factors for occurrence of PRES in pregnant women with preeclampsia were - age (<24 years), platelet count (<0.69 lacs/mm³), serum ALT (>129 IU/L) and AST (>55 IU/L), total bilirubin (>1.3 mg/dl), low haemoglobin (<8.7 g/dl) and presence of seizures. The most sensitive predicting parameters in study were serum uric acid > 5.2 mg/dl, systolic blood pressure (SBP) > 164 mm Hg, diastolic blood pressure > 100 mmHg and serum creatinine > 0.8 mg/dl (Singh *et al.*, 2021). So in pregnant women these factors should be kept in mind and screened during peripartum. But in another case report published by Zhang *et al.*, PRES can develop in pregnant women without any known risk factors like preeclampsia (Zhang *et al.*, 2022). This is similar to our patient in whom we did not detect hypertension nor any other deranged blood parameters like in the Singh *et al.*, study.

CONCLUSION

This case highlights several atypical aspects of PRES/RCVS such as absence of thunderclap headache, absence of typical underlying risk factors such as hypertension, relapse in the absence of a known precipitant. This case therefore highlights that a suspicion of PRES/RCVS should be made even in the absence of a typical background features and early therapy should be instituted to prevent complications and relapse. This case also emphasizes that PRES and RCVS possibly represent a clinicopathological spectrum with a common pathophysiological basis and further research in needed to clearly define this spectrum and measures to prevent the evolution of one to the other.

ABBREVIATIONS

PRES- Posterior Reversible Encephalopathy Syndrome
RCVS- Reversible Cerebral Vasoconstriction Syndrome

MRI- Magnetic Resonance Imaging
FLAIR- Fluid -Attenuated Inversion Recovery
DWI- Diffusion Weighted Imaging
PACNS- Primary Angiitis of the Central Nervous System
SAH- Subarachnoid Haemorrhage
BBB- Blood Brain Barrier
MRA- Magnetic Resonance Angiography
DSA- Digital Subtraction Angiography
TCH- Thunderclap Headache
LUCS- Lower Uterine Caesarean Section
BP- Blood Pressure
EEG- Electroencephalography
CSF- Cerebrospinal Fluid
ADA- Adenosine Deaminase
ANA- Antinuclear Antibody
RA- Rheumatoid Arthritis
Anti CCP- Anti Cyclic Citrullinated Peptide
SLE- Systemic Lupus Erythematosus
GCS- Glasgow Coma Scale
UMN- Upper Motor Neuron

DECLARATIONS

Ethics Approval and Consent to Participate Declaration

Ethics approval is not applicable for our case report as no separate intervention or trial was done. This is only a observational reporting.

Consent to participate was taken from the patient and her guardian - verbal and informed written consent was obtained.

Consent for Publication Declaration

Verbal and written informed consent for publication of case details including images, patient details and investigation reports was taken from the patient. Such was given willingly by her and her guardian. However, it was ensured that anonymity of the patient would be maintained in any publication.

Data Availability Declaration: Not Applicable.

Data sharing is not applicable to this article as no datasets were generated or analysed during the current study.

Competing Interests: There are no conflict of interests.

Funding: No funding was required for this case report.

Authors' Contributions

Dr Basundhara Saha(Corresponding author) was involved in data collection, patient examination and obtaining consent and also is the author of the case report.

Dr Debabrata Chakraborty provided the primary guidance in writing of the article. He edited the article and also provided authorship.

Dr Sadanand Dey was the treating neurologist of the case, and provided the data for the article.

All authors have contributed to the article and agree to their authorship in this article.

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