IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 22, Issue 9 Ser.1 (September. 2023), PP 43-44www.iosrjournals.org

A Case Report Of Atypical Posterior Reversible Encephalopathy Syndrome In A Post-Partum Patient With Malaria, Scrub Typhus And Severe Anemia

Dr. Aayushi Lal[1] , Dr. N. K. Gupta[2]
Resident[1] , Professor[2]
Department of General Medicine Pacific Institute of Medical Sciences, Udaipur, India

Abstract
Posterior reversible encephalopathy syndrome (PRES) is a neurological condition that is believed to arise from elevated blood pressure with failure of autoregulation resulting in capillary leak, vasogenicedema, and bloodbrain barrier dysfunction. It may present with diverse clinical symptoms including visual disturbance, headache, seizures and impaired consciousness. PRES classically involves bilateral parieto-occipital lobes and is usually reversible. Atypical variant of PRES includes the involvement of brainstem, basal ganglia, thalami, or periventricular white matter. A variety of clinical conditions are associated with the development of PRES, common ones include hypertensive emergency, renal disease, pre-eclampsia/eclampsia, immunosuppressive agents, sepsis, autoimmune diseases and blood transfusions. Cumulative effects of blood transfusion on blood flow, blood viscosity, endothelial dysfunction leads to blood-brain barrier dysfunction, which results into vasogenicedema and vasoconstriction despite normal systemic blood pressure, leading to blood-transfusionrelated PRES.

Key words *Pregnancy, Falciparum, Encephalopathy, PRES, Atypical PRES*

Date of Submission: 24-08-2023 Date of Acceptance: 04-09-2023

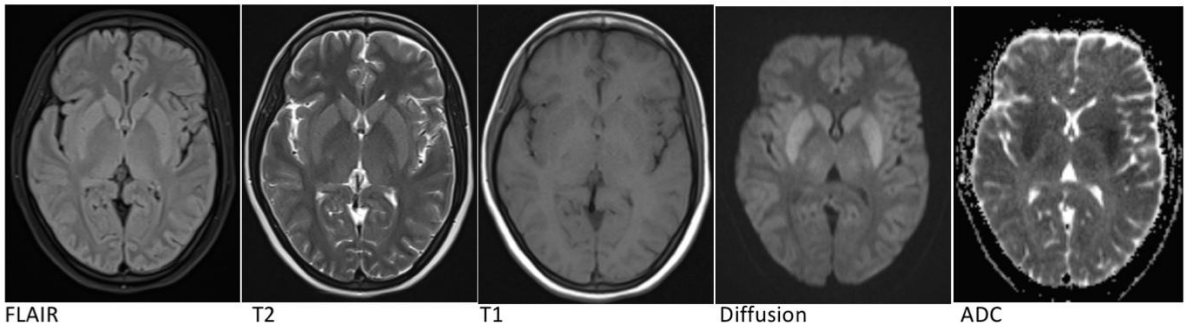
I. Case Presentation

A 34 year old female, presented to the obstetric department with the complaints of fever, generalised weakness and giddiness 10 days after Normal vaginal delivery of a healthy baby girl. She had an otherwise uneventful pregnancy. All her antenatal blood pressure recordings were within normal limits. Family history was unremarkable.

Physical examination revealed she was conscious, alert and oriented on admission with a temperature of 102 degrees F, pulse rate of 88/minute and blood pressure of 114/60 mmHg. Respiratory, cardiac and abdominal examination was within normal limits.

On evaluation, her hemoglobin was 3.0 gm% with WBCs 16,500 /cumm with 27,000/cumm platelets with Normal liver function tests along with PBF for MP showing ring stage of plasmodium falciparum and scrub typhus IgM antibodies were also positive. Hence, she was transferred to the Department of General Medicine where the initial treatment with IV Doxycycline and Artesunate was started along with 3 units of PRBC and 2 units of RDP were transfused.

On the third day of hospitalisation, the patient worsened with complaints of headache, post which she became disoriented, non-cooperative and was unable to speak but was still conscious and also complained of chest pain as well diminution of vision. Pupils were normally reactive to light and her pulse rate was 132/minute with blood pressure was 122/78 mmHg. There was no evidence of neck rigidity. Chest X-ray showed cardiomegaly and on auscultation, crepts were present bilaterally along with systolic murmurs in all 4 cardiac areas.



Further evaluation with MRI Brain revealed symmetrical areas of restricted diffusion in bilateral basal ganglia which appeared hyperintense on T2W / FLAIR images and hypointense on T1W images. No blooming on SW1 was noted, findings suggestive of atypical PRES with d/d of Viral encephalitis.

Patient was transferred to the intensive care unit and was further treated with IV steroids, IV fluids and IV antiepileptics along with antibiotics, oxygen support was given, and further transfusions were put on hold.

The patient's headache resolved, her vision improved, and she became oriented to time, place, person as well as started following commands and became cooperative. She was later transferred to the ward and continued to improve and was discharged home symptom free on the eighth day of hospitalisation.

II. Discussion

Sudden deterioration of our patient after blood transfusions and then her quick recovery supports our diagnosis of atypical PRES. We all know Immunologic as well as non-immunologic complications of blood transfusion but PRES is rarely seen post transfusion. The pathogenesis leading to transfusion-related PRES in chronically anemic patients is that chronic anemia may result in compensatory cerebral vasodilatation. The blood transfusion leads to increased blood flow as well as blood viscosity, which results in impaired hypoxic vasodilation, thus an increase in vascular resistance, and ultimately, generalized cerebral vessel constriction. Rapid increase in hematocrit levels with increased blood viscosity and release of prostaglandins, calcium, serotonin, nitric oxide, and endothelin-1 exacerbates endothelial dysfunction, which leads to blood-brain barrier dysfunction resulting into vasogenicedema and vasoconstriction despite normal systemic BP. Abrupt or acute cerebral hyperperfusion overwhelming the autoregulation of cerebral capillary perfusion pressure may result in vasogenicedema. The release of catecholamines is also suggested as a possible mechanism of vasoconstriction. Cerebral vasoconstriction can be considered the inciting event of PRES rather than endothelial dysfunction leading to cerebral vasoconstriction and subsequently PRES Along with associated factors like infections or sepsis.

References

[1]. Hinduja A. Posterior Reversible Encephalopathy Syndrome: Clinical Features And Outcome. Front Neurol. 2020;11:71. [PMC Free Article] [Pubmed] [Google Scholar]
[2]. Mitaka H, Seijo L, Motohashi K, Nakai M, Burger A. Posterior Reversible Encephalopathy Syndrome Induced By Red Blood Cell Transfusion. QJM. 2019;112:617–8. [Pubmed] [Google Scholar]
[3]. Cevallos CA, Berman BS. Posterior Reversible Encephalopathy Syndrome After Blood Transfusion. J Neurol Sci. 2016;367:245–6. [Pubmed] [Google Scholar]
[4]. Posterior Reversible Encephalopathy Syndrome, Part 1: Fundamental Imaging And Clinical Features. Bartynski WS. AJNR Am J Neuroradiol. 2008;29:1036–1042. [PMC Free Article] [Pubmed] [Google Scholar]
[5]. Posterior Reversible Encephalopathy Syndrome: Incidence Of Atypical Regions Of Involvement And Imaging Findings. Mckinney AM, Short J, Truwit CL, Mckinney ZJ, Kozak OS, Santacruz KS, Teksam M. AJR Am J Roentgenol. 2007;189:904–912. [Pubmed] [Google Scholar]
[6]. Legriel S , Schraub O , Azoulay E , Et Al . Determinants Of Recovery From Severe Posterior Reversible Encephalopathy Syndrome. Plos One 2012;7:E44534.Doi:10.1371/Journal.Pone.0044534 (Http://Dx.Doi.Org/10.1371/Journal.Pone.0044534) Pmid:Http://Www.Ncbi.Nlm.Nih.Gov/Pubmed/23024751
[7]. Servillo G, Striano P, Striano S, Et Al. Posterior Reversible Encephalopathy Syndrome (PRES) In Critically Ill Obstetric Patients. Intensive Care Med 2003;29:2323–6 [Pubmed] [Google Scholar]