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Posterior Reversible Encephalopathy Syndrome in Ischaemic Stroke with Malignant Hypertension, Chronic Kidney Disease, And Septic : A Case Report

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ABSTRACT

Posterior Reversible Encephalopathy Syndrome (PRES) is a diagnosis based on neuroimaging with the finding of vasogenic edema, especially in the occipital and parietal lobes. Some of the risk factors include malignant hypertension, chronic kidney failure, organ transplantation, autoimmune, immunosuppressant drugs, chemotherapy, and sepsis. The PRES mechanism is due to disrupted autoregulation, especially in the posterior circulation which is associated with hypertension and hypoperfusion. This will cause damage to the blood-brain barrier and vasogenic edema. We report a woman, 70 years old, with the chief complaint of loss of consciousness accompanied by headache, vomiting, and restlessness. The patient has chronic hypertension and routine hemodialysis due to chronic kidney failure. On physical examination, the patient was somnolent, malignant hypertension, and hemiparesis sinistra. Imaging examination showed vasogenic edema of the right parietooccipital lobe. The patient was treated in the intensive care unit, given intravenous dexamethasone, antihypertension, antibiotic, and routine hemodialysis. Consciousness gradually improved.

KEYWORDS: Chronic Kidney Diesease, PRES, septic, stroke.

ARTICLE DETAILS

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INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) or also known as reversible posterior leukoencephalopathy syndrome (RPLS) is a relatively fast and reversible syndrome due to malignant hypertension (most often) and not associated with hyperlipidemia. (1,2) This syndrome has symptoms including headache, nausea, vomiting, visual disturbances, and in advanced cases can cause impaired consciousness to coma and seizures. The term PRES may be used for those syndromes and should not be used to refer to chronic recurrent headaches, dizziness, epileptic seizures, TIA, or stroke, which may occur in association with an increase in blood pressure. (3,4)

Risk factors and causative factors associated with PRES include malignant hypertension, end-stage renal failure, organ transplantation, autoimmune disorders, immunosuppressant drugs, cancer chemotherapy, sepsis, preeclampsia, and eclampsia. Because most PRES patients present with clinically severe hypertension, several theories suggest that PRES is a manifestation in the spectrum of hypertensive encephalopathy. Encephalopathy can also cause extreme hypertension from several causes, including chronic kidney disease, acute glomerulonephritis, renal artery stenosis, Cushing's syndrome, pheochromocytoma, cocaine, and drugs administration such as aminophylline and phenylephrine.(5,6)

Imaging examination shows that there is vasogenic edema in the posterior area, especially in the parietooccipital lobe (although the anterior area can also be involved) so that symptoms are usually dominated from this area, such as visual disturbances, sensory disturbances, hallucinations, and cortical blindness. This disorder is often accompanied by focal or lateralized neurological symptoms due to infarction or hemorrhagic stroke. Although there is often bilateral vasogenic edema, unilateral lesions can be found. The T2 MRI examination shows an increase in intensity and the plain

head MSCT examination shows a decrease in density which was concentrated in the posterior hemisphere area. In most but not all cases, the LCS protein will increase by more than 100 mg/dl but no cellular reaction is found. (7,8)

Management of patients with PRES consists of aggressive blood pressure control. Most patients with PRES require observation in the intensive care unit. In addition, management of the underlying condition or discontinuation of the precipitating drug is the most important. (9,10,11)

CASE REPORT

A 70 year old woman came to the hospital because of loss of consciousness since 3 hours before. The patient tended to be drowsy and restless. The patient could respond to sound stimulation and strong shaking but returned to sleep again. Prior to loss of consciousness, the patient vomited five times and complained of pain throughout the head. The patient also felt that the limbs on the left side were weak. Numbness was unknown. Blurred vision and double vision were unknown, dizziness and swallowing disorder were denied. Defecation was still good. The patient could urinate a little on the diaper. Because there was no recovery of consciousness for 3 hours, the family took the patient to the hospital. The patient has had a history of diabetes mellitus since 20 years ago, hypertension since 15 years ago, routine hemodialysis since 5 years ago, and a history of stroke twice affecting the right side of the limb. The patient routinely took drugs from the several previous doctors such as trimetazidine dyhidrochloride twice a day, lercanidpine twice a day, lenagliptin/metformin 5 mg once a day, beraprost twice a day, metildopa three times a day, half tablet of carvedilol once a day.

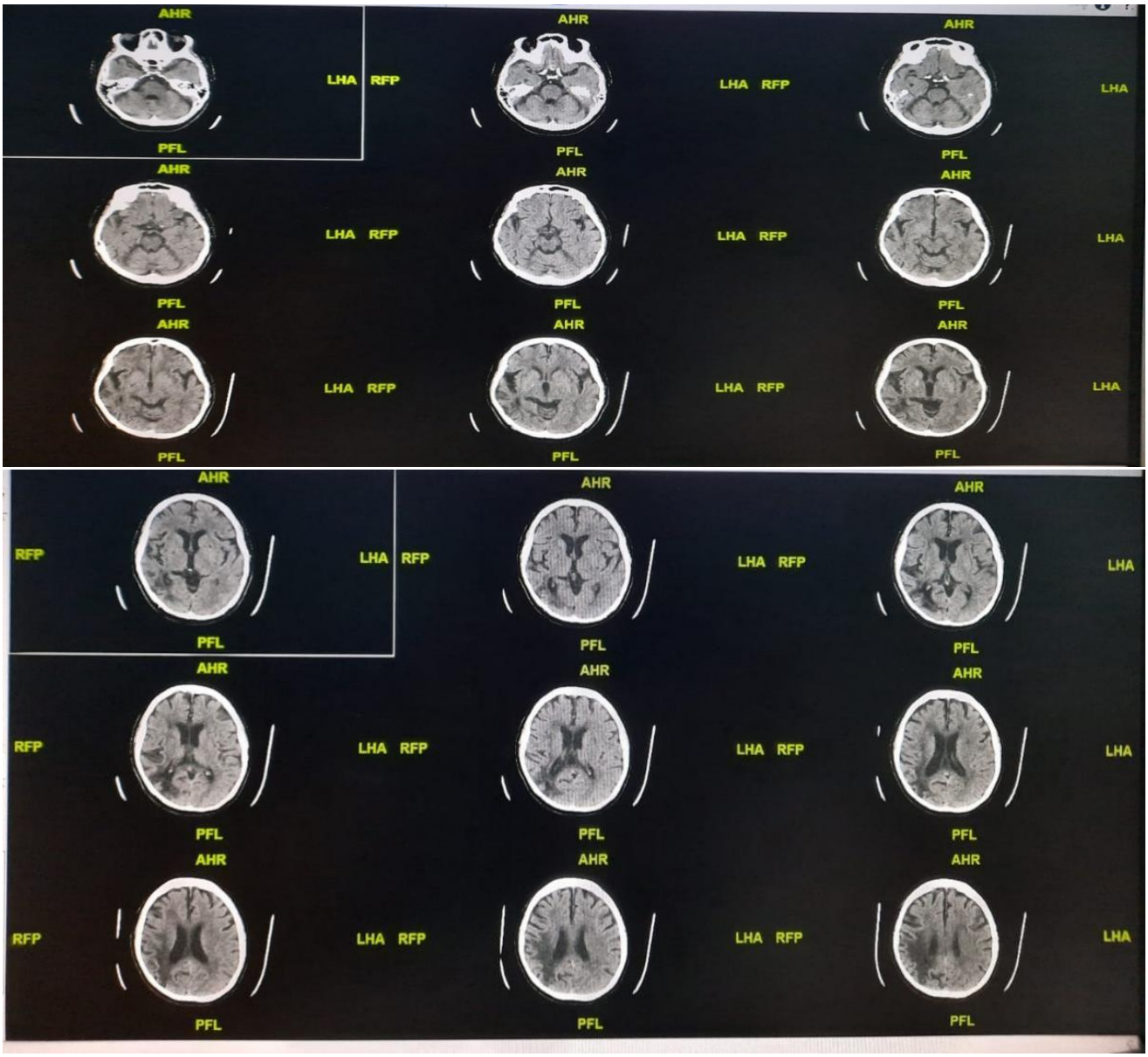

Figure 1. MSCT of the initial patient's plain head, there was an right parietooccipital lobe vasogenic edema

Physical examination showed that the patient was found somnolent, E3M6V4 and looked restless. Blood pressure was 210/120 mmHg, heart rate 110 times per minute, respiratory rate 22 times per minute, and temperature 36.7 degrees Celsius. The examination of eye movements showed that light and pupil reflexes were within normal limits, left central facial nerve parese and left central hypoglossal nerve parese, bilateral hemiparesis especially on the left side of the UMN type (right side sequele). EKG examination showed sinus tachycardia, 105 beats per minute, normoaxis. Chest X-ray examination revealed cardiomegaly. On plain head MSCT examination, there was an infarction of the right and left corona radiata, left and right interna capsule, left and right basal ganglia, and right parietooccipital lobe vasogenic edema. Laboratory results showed leukocyte count of 13,700/uL, NLR 4.66, Creatinin 8.24 mg/dL, Ureum 39.7 mg/dL, eGFR 4.5 ml/minute/body surface area, blood sugar at 294 mg/dL.

In the hospital, the neurologist did primary survey, administered 10mg loading injection of dexamethasone then continued 5 mg every 6 hours, Diltiazem intravenous syringe pump, injection of paracetamol 1 gram every 8 hours, injection of lansoprazole 30 mg every 12 hours, clopidogrel 75 milligrams every 24 hours, and oral lisinopril 10 mg every 24 hours. Then the patient was treated in the ICU and routine hemodialysis was programmed twice a week.

During the first day in the ICU, the patient was programmed to lower the blood pressure by 20 percent to 170/95 mmHg. The patient's consciousness improved and became composmentis within the first day of hospitalization. The patient did not experience vomiting but still felt headache and restlessness. On the second day of hospitalization in the ICU, the patient was programmed to lower her blood pressure by 20 percent again to 135/80 mmHg. The patient's consciousness remained composmentis, did not vomit, had minimal headache, and was not restless. On the third day of hospitalization in the ICU, the patient underwent routine hemodialysis. Because random blood sugar test and fasting blood sugars test were not controlled, the neurologist started decreasing the dose of intravenous dexamethasone to 5 mg every 8 hours, 5 mg every 12 hours on the fourth day, and 5 mg every 24 hours on the fifth day. On the sixth day the patient was not given intravenous dexamethasone. Intravenous diltiazem via syringe pump was stopped on the third day and replaced by oral antihypertensive drugs.

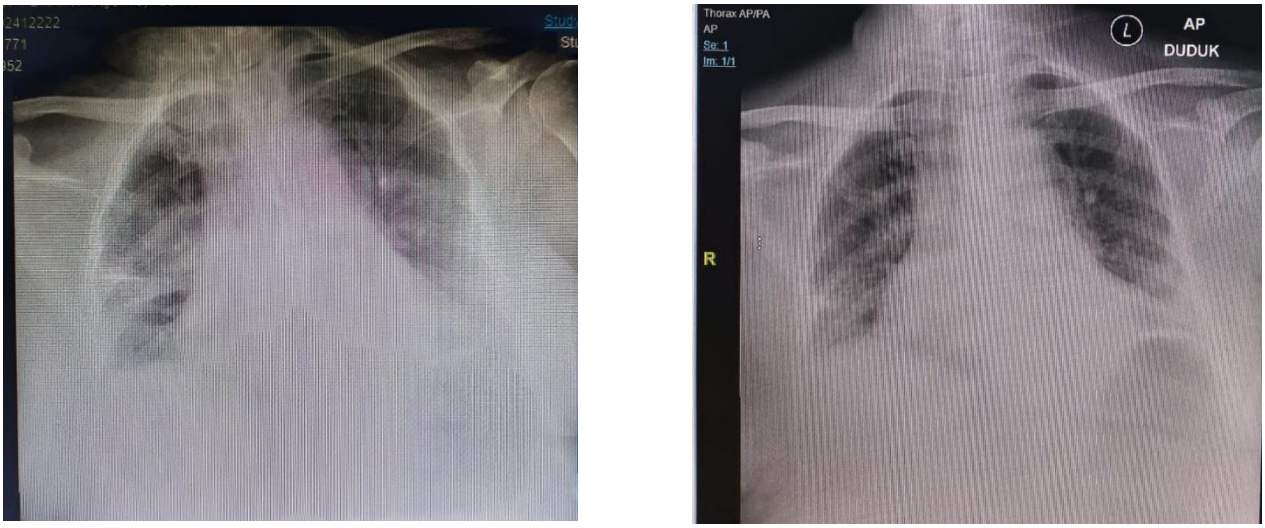

Figure (2a). First chest x-ray with pneumonia. (2b) Thorax X-ray evaluation after administration of Linezolid showed improvement

On the fifth day of hospitalization, the patient complained of shortness of breath and cough. From the physical examination, crackles were found in both lung fields. Thorax X-ray examination was carried out in patients with pneumonia. Laboratory results showed leukocytes 27,100/µL, LED 108 mm/hour, and NLR 39.22. The patient received intravenous Moxifloxacin therapy from the pulmonologist. On the seventh day, the patient experienced a repeated decrease in consciousness, tend to be drowsy with GCS E3M6V5 accompanied by headache. Laboratory test result showed a leukocyte count of 18,100/µL, ESR of 110 mm/hour, NLR of 27.95, and procalcitonin 2.47 ng/ml. The patient was diagnosed with sepsis and was added Ceftriaxone 2 grams every 24 hours (also given additional antibiotic with Moxifloxacin 400 mg every 24 hours). The patient did not show any significant clinical improvement for 5 days, so that the patient underwent the second head MSCT. The results of repeated head MSCT showed the appearance of left-sided parietooccipital vasogenic edema, while right-sided vasogenic edema of the posterior hemisphere had begun to decrease. The clinician gave intravenous linezolid and dexamethason again at a dose of 5 mg every 6 hours and tapered off in the following days. The patient showed improvement.

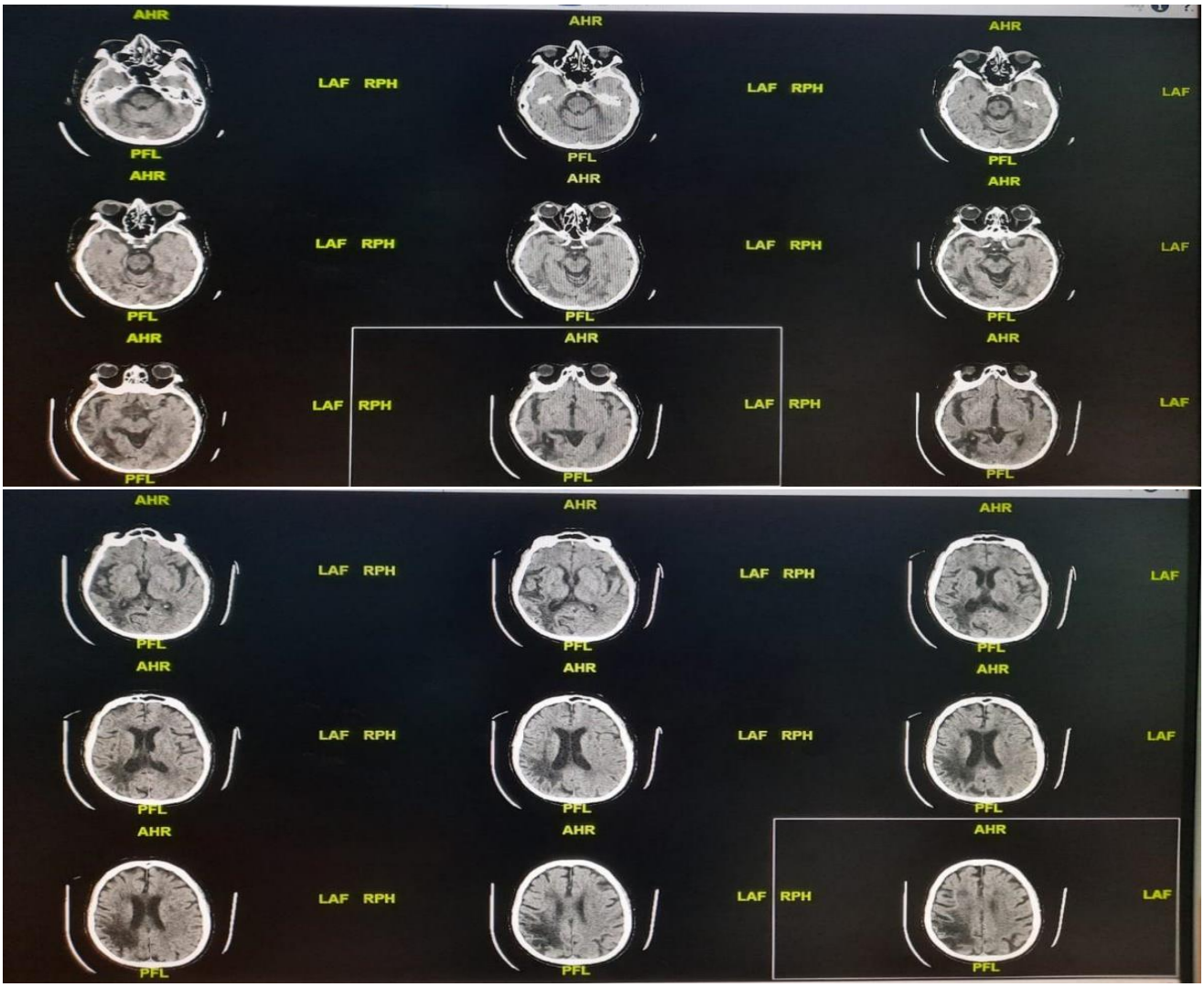

Figure 3. Plain head MSCT was repeated when the patient lost consciousness again. There was vasogenic edema in the billateral parietooccipitocerebellar lobe

DISCUSSION

The patient was diagnosed with Posterior Reversible Encephalopathy Syndrome from anamnesis, physical examination and supporting examinations.From the anamnesis, there were decreased consciousness, headache, vomiting, and restlessness as signs of increased intracranial pressure. In PRES, we get a imaging of vasogenic edema which is the result of damage to the blood-brain barrier. This is triggered by disruption of autoregulation especially in the posterior circulation which is associated with hypertension and tissue hypoperfusion. In addition, PRES is also associated with vascular endothelial damage and dysfunction. Brain edema is the result of active exocytosis of water, not simply passive leakage from blood vessels under high pressure. (12,13)

The patient's vital signs showed malignant hypertension, one of the triggers for PRES. However, patients with PRES can also occur in normotensive patients who experience a sudden increase in blood pressure. In addition, this patient has other causative factors such as end-stage chronic renal failure and sepsis. On physical examination, there were left central facial nerve paresis and left central hypoglossal nerve paresis, and left-sided severe bilateral hemiparesis of the UMN type due to mass effect of vasogenic edema located in the right parietooccipital lobe against the right-sided pyramidal tract. Meanwhile, the left side of the pyramidal tract is the sequel to the previous stroke. PRES can often occur together with infarction or hemorrhagic stroke as in this patient because of the process of fibrinoid necrosis of the arteriolar and capillary walls and the occlusion of the lumen of blood vessels by fibrin thrombi. (14,15)

Plain head MSCT examination showed a decrease in density concentrated in the white matter area of the posterior hemisphere with the impression of vasogenic edema in the right parietooccipital lobe accompanied by multiple lacunar infarction lesions. Although PRES predominates in the posterior area, PRES can also involve more anterior areas. These imaging characteristics are the result of fluid accumulation but unlike cases of trauma, large stroke, and neoplasm, PRES has a smaller mass effect. In patients with hypertensive encephalopathy and eclampsia, it can even cause subarachnoid hemorrhage that is not caused by a

ruptured aneurysm. Although the headache resulting from subarachnoid hemorrhage due to PRES is less severe than aneurysm rupture. (16,17)

The patient was observed in the intensive care unit and administered intravenous dexamethasone and intravenous diltiazem syringe pump. By administering dexamethasone, it is hoped that it will repair the damage to the blood-brain barrier so that the mass effect does not arise due to vasogenic edema. However, administration of dexamethasone will interfere with the patient's blood sugar regulation so that the dose should be reduced when clinical improvement occurs. With uncontrolled blood sugar results (> 250 mg/dl), it will be difficult for the patient to be programmed to mobilize so that it will trigger sepsis pneumonia. Apart from that, the effect of high blood sugar itself will cause various kinds of serious infections. For this patient, other causative factors were managed, namely routine hemodialysis for indications of end-stage chronic renal failure and administration of antibiotics according to blood culture test in the management of sepsis.(18,19,20)

The relationship may be supported by the effects of sepsis on the vascular endothelium, with the impairment of cerebral autoregulatory mechanisms representing the pathophysiological process of PRES, while others demonstrated that it binds to the angiotensin-converting enzyme 2 (ACE2) receptor, which is highly expressed in neurons and glial cells. The alteration of the endothelium may be the result of hyperpermeability of the endothelium: caused by inflammation and disruption of junctional proteins due to cytokine/chemokine storm and adhesion molecules.(20,21)

Management of malignant hypertension in PRES must be done carefully to target on reducing MAP by 20% so that it is still considered quite safe. With adequate antihypertensive administration, clinical improvement will usually occur within one or two days. Treatment for malignant hypertension other than intravenous diltiazem are intravenous sodium nitroprusside, calcium channel blockers such as sublingual nifedipine, or beta-adrenergic blockers such as intravenous labetalol. In cases of pre-eclampsia or eclampsia, intravenous magnesium sulfate can be given. Longer-acting antihypertensive agents such as ACE inhibitors and calcium channel blockers should be given after the management of malignant hypertension has improved to control daily blood pressure. (22,23,24)

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This article is original and there is no conflict of interest from the author.

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REFERENCES

I. Ando Y, Ono Y, Sano A, Fujita N, Ono S. Posterior Reversible Encephalopathy Syndrome: A Review of the Literature. Intern Med. 2022 Jan 15;61(2):135-141. doi: 10.2169/internalmedicine.7520-21. Epub 2021 Jul 17.

II. Hinduja A. Posterior Reversible Encephalopathy Syndrome: Clinical Features and Outcome. Front Neurol. 2020 Feb 14;11:71. doi: 10.3389/fneur.2020.00071.

III. Adetiloye AO, Valencia Manrique J, Victoria A, Haider H, Al-Juboori MT. Posterior Reversible Encephalopathy Syndrome Presenting Atypically as a Non-Convulsive Seizure. Am J Case Rep. 2021 Nov 5;22:e933667. doi: 10.12659/AJCR.933667.

IV. Fischer M, Schmutzhard E. Posterior reversible encephalopathy syndrome. J Neurol. 2017 Aug;264(8):1608-1616. doi: 10.1007/s00415-016-8377-8. Epub 2017 Jan 4.

V. How J, Blattner M, Fowler S, Wang-Gillam A, Schindler SE. Chemotherapy-associated Posterior Reversible Encephalopathy Syndrome: A Case Report and Review of the Literature. Neurologist. 2016 Nov;21(6):112-117. doi: 10.1097/NRL.0000000000000105.

VI. Singer S, Grommes C, Reiner AS, Rosenblum MK, DeAngelis LM. Posterior Reversible Encephalopathy Syndrome in Patients With Cancer. Oncologist. 2015 Jul;20(7):806-11. doi: 10.1634/theoncologist.2014-0149. Epub 2015 Jun 1.

VII. Chen TH. Childhood Posterior Reversible Encephalopathy Syndrome: Clinicoradiological Characteristics, Managements, and Outcome. Front Pediatr. 2020 Sep 11;8:585. doi: 10.3389/fped.2020.00585.

VIII. Koksel Y, McKinney AM. Potentially Reversible and Recognizable Acute Encephalopathic Syndromes: Disease Categorization and MRI Appearances. AJNR Am J Neuroradiol. 2020 Aug;41(8):1328-1338. doi: 10.3174/ajnr.A6634. Epub 2020 Jul 2.

IX. Algahtani H, Algahtani A, Aldarmahi A, Hmoud M, Marzuk Y, Shirah B. Posterior Reversible Encephalopathy Syndrome: Local Experience From Saudi Arabia. Neurohospitalist. 2017 Jan;7(1):24-29. doi: 10.1177/1941874416665762. Epub 2016 Aug 31

X. Nakamura Y, Sugino M, Tsukahara A, Nakazawa H, Yamamoto N, Arawaka S. Posterior reversible encephalopathy syndrome with extensive cytotoxic edema after blood transfusion: a case

report and literature review. BMC Neurol. 2018 Nov 12;18(1):190. doi: 10.1186/s12883-018-1194-1.

XI. Ansari B, Saadatnia M. Prevalence and Risk Factors of Posterior Reversible Encephalopathy Syndrome in Isfahan, Iran. Adv Biomed Res. 2021 Dec 25;10:53. doi: 10.4103/abr.abr_243_19.

XII. Ou S, Xia L, Wang L, Xia L, Zhou Q, Pan S. Posterior Reversible Encephalopathy Syndrome With Isolated Involving Infratentorial Structures. Front Neurol. 2018 Oct 9;9:843. doi: 10.3389/fneur.2018.00843.

XIII. Cheng X, Li J, Lan Y, Liu J, Chen S, Lu G. Cerebrovascular Disease in the Setting of Posterior Reversible Encephalopathy Syndrome. Front Neurol. 2021 Nov 17;12:765333. doi: 10.3389/fneur.2021.765333.

XIV. Alshami A, Al-Bayati A, Douedi S, Hossain MA, Patel S, Asif A. Clinical characteristics and outcomes of patients admitted to hospitals for posterior reversible encephalopathy syndrome: a retrospective cohort study. BMC Neurol. 2021 Mar 9;21(1):107. doi: 10.1186/s12883-021-02143-6.

XV. Seitz A, Parauda SC, Salehi Omran S, Schweitzer AD, Liberman AL, Murthy SB, Merkler AE, Navi BB, Iadecola C, Kamel H, Zhang C, Parikh NS. Long-term risk of seizure after posterior reversible encephalopathy syndrome. Ann Clin Transl Neurol. 2023 Apr;10(4):610-618. doi: 10.1002/acn3.51748. Epub 2023 Feb 22.

XVI. Bansal S, Bansal R, Goyal MK, Takkar A, Singh R, Singh P, Lal V. Clinical, Etiological and Imaging Profile of Posterior Reversible Encephalopathy Syndrome: A Prospective and Follow-Up Study. Ann Indian Acad Neurol. 2020 Mar-Apr;23(2):182-188. doi: 10.4103/aian.AIAN_379_18. Epub 2020 Feb 25.

XVII. Çamlıdağ İ, Cho YJ, Park M, Lee SK. Atypical Unilateral Posterior Reversible Encephalopathy Syndrome Mimicking a Middle Cerebral Artery Infarction. Korean J Radiol. 2015 Sep-Oct;16(5):1104-8. doi: 10.3348/kjr.2015.16.5.1104. Epub 2015 Aug 21.

XVIII. Ali K, Karan A, Biswah S, Teelucksingh S, Mohammed NB. Posterior Reversible Encephalopathy Syndrome: Tips for Diagnosis and Treatment. Cureus. 2021 Mar 24;13(3):e14087. doi: 10.7759/cureus.14087.

XIX. Shieh A, Darro N. Recurrent Posterior Reversible Encephalopathy Syndrome in an Adolescent Boy with End-Stage Renal Disease. Case Rep Pediatr. 2021 Feb 16;2021:6675454. doi: 10.1155/2021/6675454.

XX. Garner O, Ramirez A, Iardino A. A case of posterior reversible encephalopathy syndrome associated with sepsis. BMJ Case Rep. 2018 Jul 10;2018:bcr2018225128. doi: 10.1136/bcr-2018-225128.

XXI. Iftikhar S, Rehman AU, Ameer MZ, Nawaz A, Aemaz Ur Rehman M, Farooq H, Asmar A, Ebaad Ur Rehman M. The association of posterior reversible encephalopathy syndrome with COVID-19: A systematic review. Ann Med Surg (Lond). 2021 Dec;72:103080. doi: 10.1016/j.amsu.2021.103080. Epub 2021 Nov 20.

XXII. Parikh NS, Schweitzer AD, Young RJ, Giambrone AE, Lyo J, Karimi S, Knobel A, Gupta A, Navi BB. Corticosteroid therapy and severity of vasogenic edema in posterior reversible encephalopathy syndrome. J Neurol Sci. 2017 Sep 15;380:11-15. doi: 10.1016/j.jns.2017.06.044. Epub 2017 Jun 29.

XXIII. Chen Z, Zhang G, Lerner A, Wang AH, Gao B, Liu J. Risk factors for poor outcome in posterior reversible encephalopathy syndrome: systematic review and meta-analysis. Quant Imaging Med Surg. 2018 May;8(4):421-432. doi: 10.21037/qims.2018.05.07.

XXIV. Thompson RJ, Sharp B, Pothof J, Hamedani A. Posterior reversible encephalopathy syndrome in the emergency department: case series and literature review. West J Emerg Med. 2015 Jan;16(1):5-10. doi: 10.5811/westjem.2014.12.24126. Epub 2015 Jan 5.