Case Report

Indian Journal of Emergency Medicine
Volume 3 Number 1, January - June 2017
DOI: http://dx.doi.org/10.21088/ijem.2395.311X.3117.22

Hypertension-Induced Posterior Reversible Encephalopathy Syndrome as the Presentation of Progressive Bilateral Renal Artery Stenosis

Monilkumar Patel[1], Kishalay Datta[2], Rigenjyoti Kalita[3], Anita Rawat[4], Hilal Ahmad Yatoo[4], Jitesh Bhandarkar[1]

Author's Affiliation: [1]DNB Resident [2]Principal Consultant & HOD [3]MEM Resident [4]Attending Consultant, Emergency Medicine, Max Hospital, Shalimar Bagh, New Delhi-110088, India.

Corresponding Author: Monilkumar Patel, DNB Resident, Department of Emergency Medicine, Max Hospital, Shalimar Bagh, New Delhi-110088, India.
E-mail: monilpatel593@gmail.com

Received on 02.01.2017,
Accepted on 23.01.2017

Abstract

Posterior reversible encephalopathy syndrome (PRES) is characterized clinically by headache, altered mental status, visual loss, and seizures. PRES is associated with neuroradiological findings characterized by white matter abnormalities, predominantly in the parieto-occipital regions of the brain. PRES is most often described in cases of hypertensive encephalopathy, eclampsia, renal failure, and immunosuppressive or anticancer therapy. We report a case of PRES associated with severe hypertension in the setting of a progressive renovascular hypertension from bilateral atherosclerotic renal artery stenosis. The pathogenesis of PRES is discussed and the importance of a prompt diagnosis and treatment is emphasized.

Keywords: Reversible Encephalopathy; Renovascular Hypertension; Magnetic Resonance Imaging.

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a disorder predominantly affecting the white matter in the posterior portion of the cerebral hemispheres. Typical symptoms of PRES include altered mental status (encephalopathy), seizure, and headache. Less commonly there may be visual disturbances, focal neurologic sign, and status epilepticus. PRES is most often described in cases of hypertensive encephalopathy, preeclampsia and eclampsia, renal failure, or following immunosuppressive or anticancer therapy. Other reported causes include autoimmune diseases, thrombotic thrombocytopenic Purpura, acute intermittent porphyria, organ transplantation, hypercalcemia, and sepsis . In most cases, an abrupt rise in blood pressure is thought to be the cause of PRES by means of acute disruption of the blood brain barrier.

Magnetic resonance imaging (MRI) shows diffuse signal abnormalities mainly involving the subcortical white matter in the parieto-occipital regions of the brain; the temporal and frontal lobes, cerebellum, basal ganglia, and brainstem may also be involved. Renal artery stenosis, from either fibrodysplasia or atherosclerosis, is a common cause of severe or resistant Posterior reversible encephalopathy syndrome and renovascular hypertension.

Case Report

23 Year Old Female was brought to emergency complaining of unconsciousness and drowsy state since the morning of presentation. As told by her mother she was found to have abnormal body movements and up rolling of eyes followed by decreased responsiveness and vomiting. There is no history of recent fever, head trauma, chest pain, seizure disorder or any other neurological issues. She also does not have any past medical or surgical history. On arrival to the Emergency, her vital parameters were Pulse: 160 / Min; BP: 210 / 120 mm Hg; Capillary blood glucose: 181mg / dl; Temperature: 98 F; SpO2:94% on Room Air; Respiratory rate: 20 / min; Cardiac Monitor showed Sinus Tachycardia.

On neurological examination, patient was found to be drowsy and disoriented not following commands or answering questions appropriately. Glasgow coma score was recorded to be E2V3M5. Pupils were unequal and dilated with sluggish reaction to light. Her visual acuity reduced to finger

movements and perception of light. On Fundoscopy: Optic Discs blurred margin, Pale Retina with Splinter Hemorrhages, Temporal Retinal Detachment were observed.

There were spontaneous limb movements bilaterally; left plantar response was equivocal whereas, right planter was up going. The rest of the neurological examination was limited, as the patient was not following commands consistently. All other systemic examinations were essentially unremarkable.

Her initial blood reports from Emergency were as follows:

Hb: 13.3 Gm / dl; TC(WBC): 28,300; Creatinine:1.76; serum sodium:143.1; serum potassium:3.8. Renal and Liver function tests are within normal limits. RA Factor / ANA / ANCA/ VMA were all negative. Arterial blood gas report showed: pH : 7.519, PO2 : 62.0, PCO2 : 32.7, HCO3 :26 sodium : 145.3, K : 3.15, CL: 108, GLU: 218, LAC:0.75. Bedside urine pregnancy test and toxicology screen were negative. CT scan of brain was done which revealed diffuse cerebral oedema.

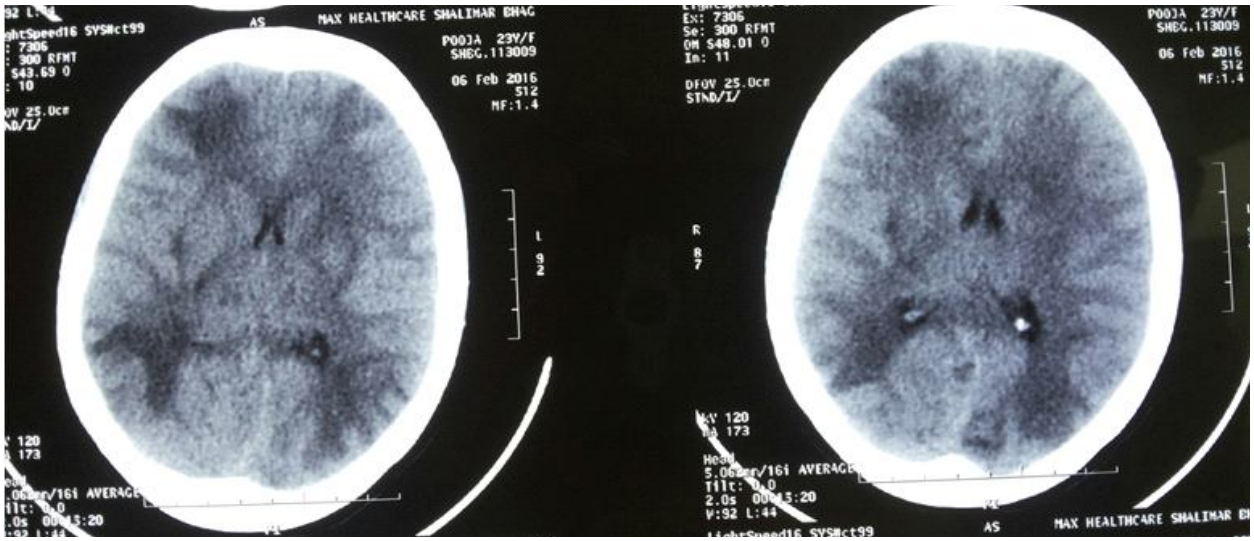

Fig. 1: NCCT Head Done on admission shows Diffuse white matter edema

Provisional Diagnosis in the Emergency department was Hypertensive Encephalopathy / Intra cranial haemorrhage / Seizure disorder or CVA.

Patient was resuscitated in Emergency department and her blood pressure was managed with intravenous antihypertensive. After an hour, BP was 170/100mm Hg and she was admitted under a Neurologist in ICU for further management. After admission, MR angiogram of brain was done which revealed diffuse vascular constriction in the MCA & PCA Territory suggestive of posterior reversible encephalopathy syndrome. Patient's consciousness levels improved after adequate control of her blood pressure on the second day of her admission. Ophthalmological examination revealed cortical blindness which improved with conservative management. CECT Abdomen + Renal CT Angiogram revealed complete occlusion of right renal artery due to thrombosis & left renal artery showed stenosis of 80-90%. Left kidney showed 23% Split Function and viable on DTPA scan. Left renal artery angioplasty was done. Patient's vision improved on subsequent days and repeat CT Brain showed improvement in Comparison to previous CT scan. Patient was discharged home in a stable condition.

Fig. 2: CT Renal Angiography : Right Renal Artery shows complete thrombosis of its origin Extending over 9 mm long segment. Left Renal Artery shows 80-90% Stenosis over 10 mm segment.

Discussion

We reported a case of PRES in the setting of a severe renovascular hypertension from bilateral atherosclerotic renal artery stenosis. Renovascular hypertension represents one of the most frequent forms of secondary hypertension.

- It is important to recognize the signs and symptoms of PRES as The management of this condition depends on the etiology and should be initiated in a timely manner.
- The treatment of the underlying cause is typically sufficient to reverse this condition. If treatment got delayed, this condition can lead to irreversible brain insult or prolonged brain insult can leads to irreversible brain infarctions. Brain CT perfusion can play an important role in the diagnosis.
- The MAP should be reduced quickly but with caution in the cases of hypertensive PRES
- Posterior reversible encephalopathy syndrome is a relatively rare syndrome that sometimes presents as a stroke mimic. As such, it is important for the emergency physician to recognize.
- Urgent recognition and early initiation of management of this condition are imperative as it directly impacts the neurological outcome.

References

1. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, WangA, Pessin MS, Lamy C, Mas JL, Caplan LR. A reversible posterior leukoencephalopathy syndrome. N Engl J Med 1996; 334:494-500.
2. Garg RK. Posterior leukoencephalopathy syndrome. Postgrad Med J 2001; 77:24-8.
3. Kastrup O, Maschke M, Wanke I, Diener HC. Posterior reversible encephalopathy syndrome due to severe hypercalcemia. J Neurol 2002; 249:1563-6.
4. Fugate JE, Claassen DO, Cloft HJ, Kallmes DF, Kozak OS, Rabinstein AA. Posterior reversible encephalopathy syndrome: associated clinical and radiologic findings. Mayo Clin Proc 2010; 85:427-32.
5. Ay H, Buonanno FS, Schaefer PW, Le DA, Wang B, Gonzalez RG, Koroshetz WJ. Posterior leukoencephalopathy without severe hypertension: utility of diffusion-weighted MRI. Neurology 1998; 51:1369-76.
6. Dworkin LD, Cooper CJ. Renal artery stenosis. N Engl J Med 2009; 361:1972-8.
7. Davis RP, Pearce JD, Craven TE, Moore PS, Edwards MS, Godshall CJ, Hansen KJ. Atherosclerotic renovascular disease among hypertensive adults. J Vasc Surg 2009; 50:564-70.
8. Soulez G, Oliva VL, Turpin S, Lambert R, Nicolet V, Therasse E. Imaging of renovascular hypertension: respective values of renal scintigraphy, renal doppler US, and MR angiography. Radiographics 2000; 20: 1355-68.