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## A Case Of PRES Secondary To Renal Artery Stenosis Induced Hypertension.

**Manikandan Palaniappan\*, Venkatraman Indiran, and Ramchandraprasad.**

Department of radio-diagnosis, SreeBalaji Medical college and Hospital, No.7, Works road, Chromepet, Chennai, Tamil Nadu, India.

### ABSTRACT

Posterior reversible encephalopathy syndrome (PRES) is a condition that occurs secondary to the inability of posterior circulation to auto-regulate in response to acute changes in blood pressure with resultant hyperperfusion and vasogenicoedema, in the parieto-occipital regions. This presentation is to emphasize that renal artery stenosis, a cause of hypertension in young individuals may present as PRES. A 15 year old girl presented with head ache, giddiness and an episode of loss of consciousness for six minutes. On examination all vital parameters were normal except high blood pressure. CT Brain, MRI Brain, ultrasound abdomen and CECT abdomen was done. CT Brain showed mild diffuse cerebral edema with vasogenic edema in the bilateral parietal-occipital lobes. MRI Brain showed gyriform and sub-cortical T2 hyperintense signal change in bilateral posterior parietal and left occipital lobes. Gyriiform T2 hyperintense signal in parasagittal region in left superior frontal region with no significant restricted diffusion. USG abdomen showed contracted right kidney. Renal Doppler showed features of renal artery stenosis. CECT Abdomen showed Focal short segment high grade stenosis in juxtahilar portion of right renal artery. Diagnosis of PRES secondary to renal artery stenosis induced hypertension was made. Patient refused any surgical interventions. She was started on oral anti-hypertensive. Patient responded well to medications. Here we emphasize that Renal artery stenosis can be a cause in young patients with hypertension. While evaluating hypertensive patients with neurological symptoms like giddiness and loss of consciousness, Posterior reversible encephalopathy syndrome should be considered as a differential.

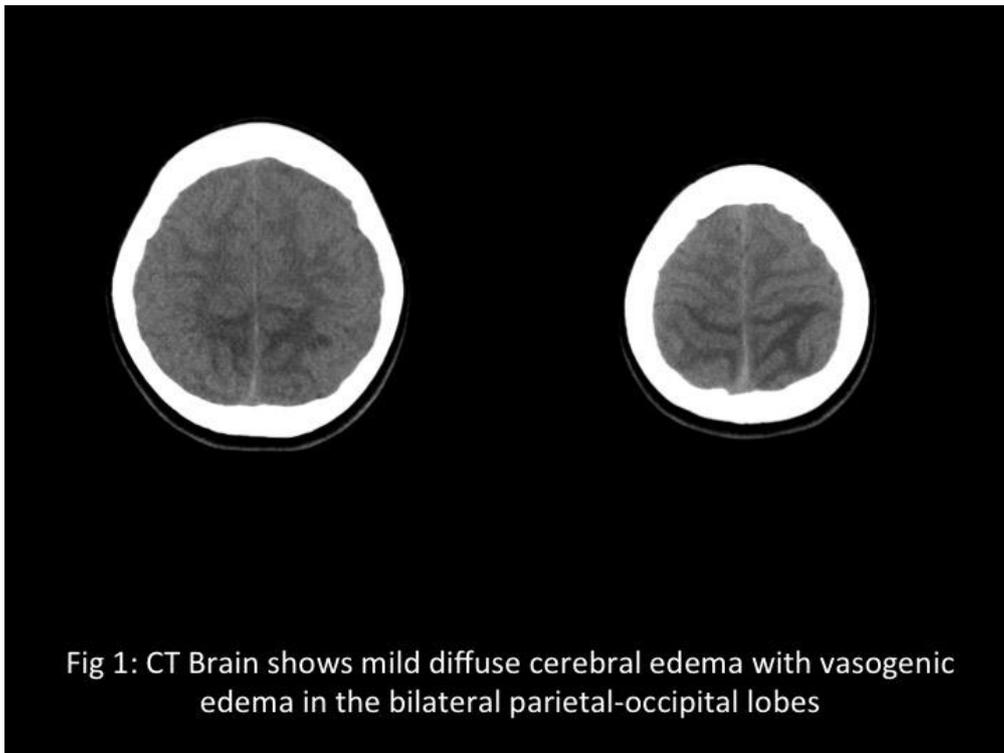
**Keywords:** Posterior Reversible Encephalopathy Syndrome (Pres), Renal Artery Stenosis, Hypertension, Pres

*\*Corresponding author*

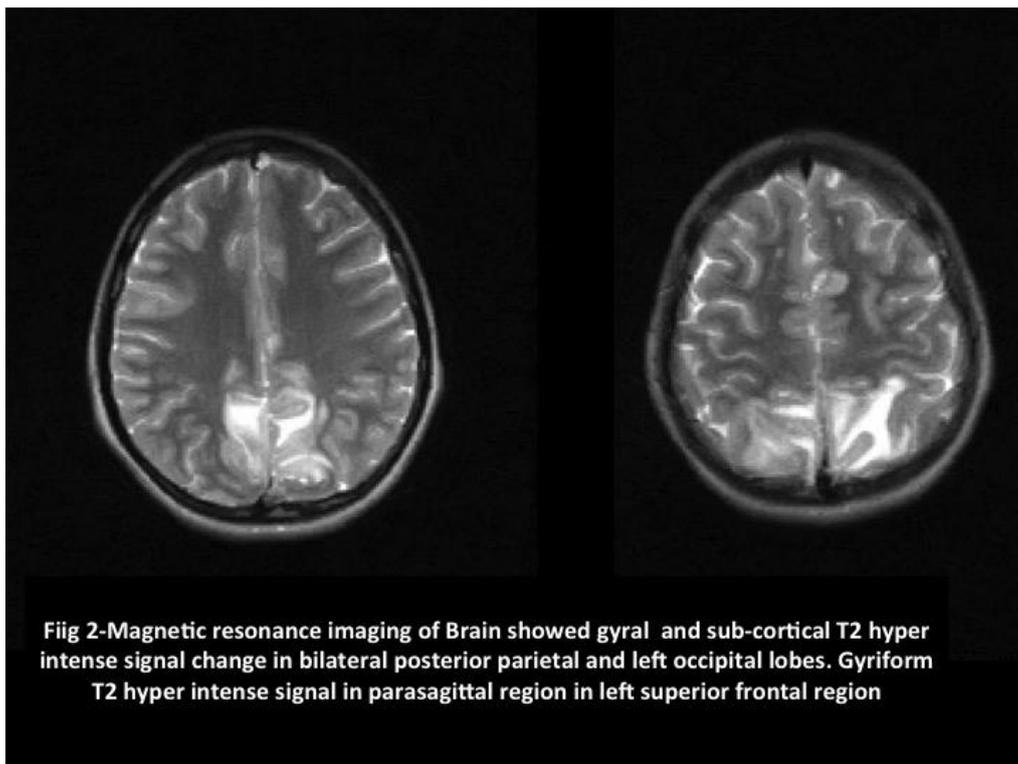
## INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) is a condition that occurs secondary to the inability of posterior circulation to auto-regulate in response to acute changes in blood pressure with resultant hyperperfusion and vasogenicoedema, in the parieto-occipital regions. PRES predominantly affect the sub cortical and deep white matter of posterior parieto-occipital lobes. Temporal lobe, frontal lobe, cerebellum, basal ganglia and brain stem can also be involved [1] [2]. Presenting symptoms include headache, seizure, decreased alertness, altered mental status. Predisposing conditions include hypertensive encephalopathy, pre-eclampsia, renal failure, chronic immune suppressive and anti cancer therapy [1]. Less common predisposition seen in autoimmune diseases, thrombotic thrombocytopenic purpura, human immune deficiency virus syndrome, acute intermittent porphyria, organ transplantation and sepsis [1]. Increased vascular permeability leading to disruption of Blood Brain Barrier (BBB) and vasogenic edema is thought to be the pathogenesis behind PRES. PRES in normotensive patients have also been reported [3], [5]. PRES is a reversible condition only when treated early. Delayed treatment can lead to permanent cerebral injury [1], [2]. Renal artery stenosis is a common cause of hypertension in young individuals. Herein we present a case of PRES secondary to renal artery stenosis induced hypertension.

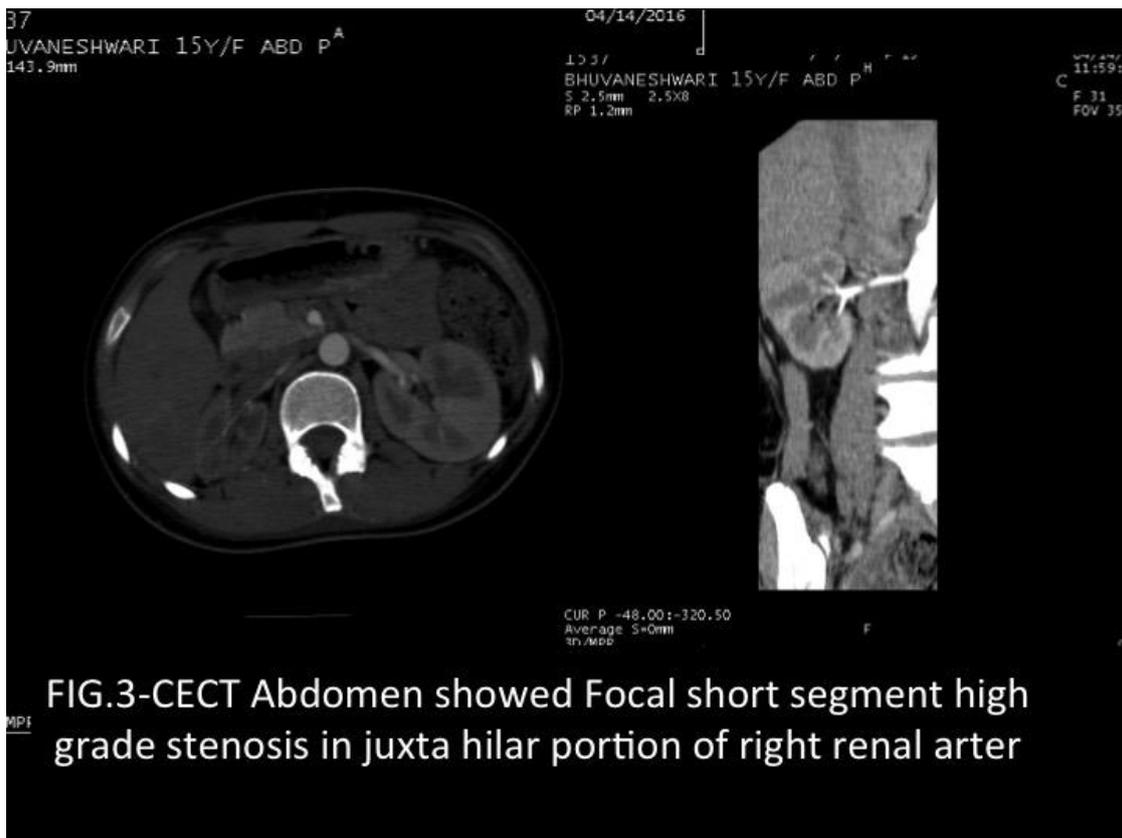
A 15 year old girl presented with headache, giddiness and an episode of sudden loss of consciousness which lasted for five minutes. On examination all parameters were within normal limits except for elevated blood pressure of 190/110 mm of hg. Patient was referred to department of Radiodiagnosis for non-contrast computed tomography which showed features of diffuse cerebral edema and vasogenic edema involving bilateral parieto-occipital lobe (Fig 1). Magnetic resonance imaging of Brain showed gyral and sub-cortical T2 hyper intense signal change in bilateral posterior parietal and left occipital lobes. Gyriiform T2 hyper intense signal in parasagittal region in left superior frontal region with no significant restricted diffusion (Fig 2). As the posterior circulation territory was involved PRES was considered as provisional diagnosis.



To evaluate the cause of hypertension USG abdomen was done, which showed contracted right kidney. Renal Doppler showed features of renal artery stenosis. CECT Abdomen showed Focal short segment high grade stenosis in juxtahilar portion of right renal artery (Fig 3). With clinical and radiological evidence diagnosis of PRES secondary to renal artery induced hypertension was made. Patient was explained about various treatment options available. As patient was not willing for any invasive / surgical procedure, she was treated conservatively with oral anti-hypertensive. On follow-up patient's blood pressure were well controlled.



**Fig 2-Magnetic resonance imaging of Brain showed gyral and sub-cortical T2 hyper intense signal change in bilateral posterior parietal and left occipital lobes. Gyriform T2 hyper intense signal in parasagittal region in left superior frontal region**



**FIG.3-CECT Abdomen showed Focal short segment high grade stenosis in juxta hilar portion of right renal arter**

### DISCUSSION

Renovascular hypertension is frequently encountered forms of secondary hypertension. Atherosclerotic renal artery stenosis, and fibromuscular dysplasia are more common cause of reno-vascular hypertension [6]. ~20 % can develop anatomical progression of renal artery stenosis[7]. Hemodynamically

significant renal artery stenosis usually results in severe and refractory hypertension or progressive renal insufficiency; clinical criteria for suspecting renovascular hypertension have been reported [8]. In this case, the abrupt onset of PRES was clearly caused by progression of a silent right renal artery stenosis to a hemodynamically significant status.

PRES is a reversible syndrome characterized by rapid-onset neurological changes [1]. Symptoms include headache, altered consciousness, nausea and vomiting, seizures, and visual symptoms including cortical blindness; focal or lateralizing neurological signs may also occur. PRES occur more commonly in severe hypertension. But PRES is reported in mild hypertension and in normo-tensive patients also [5]. Less understood pathogenesis of PRES include rapid increase in blood pressure may exceed the upper limit of cerebral autoregulation with abrupt dilation of cerebral arterioles resulting in interstitial extravasation of serum protein and fluid resulting in vasogenic edema. Vasogenic edema is mainly detectable in the white matter of the occipito-parietal regions, but may also involve brain stem, cerebellum, and basal ganglia [1]. Vasogenic edema is typically encountered in PRES, resulting probably from failure of the blood–brain barrier leading to water extravasation in the interstitial spaces and is considered a reversible pathologic situation as confirmed by reversal of symptoms in subsequent follow up. Although PRES is reversible, delay in initiation of treatment can lead to irreversible brain damage [2]. Our case is noteworthy as renal artery stenosis can be a common cause among young individual who present with hypertension. Early diagnosis and treatment can prevent irreversible damage to brain.

In conclusion, this case is a PRES caused by renal artery stenosis induced hypertension. Here we emphasize that Renal artery stenosis can be a cause in young patients with hypertension. While evaluating hypertensive patients with neurological symptoms like giddiness and loss of consciousness, Posterior reversible encephalopathy syndrome should be considered as a differential.

#### REFERENCES

- [1] Hinchey, J., Chaves, C., Appignani, B., Breen, J., Pao, L., Wang, A., Pessin, M.S., Lamy, C., Mas, J.L., and Caplan, L.R. A reversible posterior leukoencephalopathy syndrome. *N Engl J Med.* 1996; 334: 494–500
- [2] Garg, R.K. Posterior leukoencephalopathy syndrome. *Postgrad Med J.* 2001; 77: 24–28
- [3] Kastrup, O., Maschke, M., Wanke, I., and Diener, H.C. Posterior reversible encephalopathy syndrome due to severe hypercalcemia. *J Neurol.* 2002; 249: 1563–1566
- [4] Fugate, J.E., Claassen, D.O., Cloft, H.J., Kallmes, D.F., Kozak, O.S., and Rabinstein, A.A. Posterior reversible encephalopathy syndrome: associated clinical and radiologic findings. *Mayo Clin Proc.* 2010; 85: 427–432
- [5] Ay, H., Buonanno, F.S., Schaefer, P.W., Le, D.A., Wang, B., Gonzalez, R.G., and Koroshetz, W.J. Posterior leukoencephalopathy without severe hypertension: utility of diffusion-weighted MRI. *Neurology.* 1998; 51: 1369–1376
- [6] Dworkin, L.D. and Cooper, C.J. Renal artery stenosis. *N Engl J Med.* 2009; 361: 1972–1978
- [7] Davis, R.P., Pearce, J.D., Craven, T.E., Moore, P.S., Edwards, M.S., Godshall, C.J., and Hansen, K.J. Atherosclerotic renovascular disease among hypertensive adults. *J Vasc Surg.* 2009; 50: 564–570
- [8] Soulez, G., Oliva, V.L., Turpin, S., Lambert, R., Nicolet, V., and Therasse, E. Imaging of renovascular hypertension: respective values of renal scintigraphy, renal doppler US, and MR angiography. *Radiographics.* 2000; 20: 1355–1368
- [9] Doelken, M., Lanz, S., Rennert, J., Alibek, S., Richter, G., and Doerfler, A. Differentiation of cytotoxic and vasogenic edema in a patient with reversible posterior leukoencephalopathy syndrome using diffusion-weighted MRI. *Diagn Interv Radiol.* 2007; 13: 125–128
- [10] Benziada-Boudour, A., Schmitt, E., Kremer, S., Foscolo, S., Rivière, A.S., Tisserand, M., Boudour, A., and Bracard, S. Posterior reversible encephalopathy syndrome: a case of unusual diffusion-weighted MR images. *J Neuroradiol.* 2009; 36: 102–105
- [11] Parker, R., Ostermann, M., Reidy, J., and Goldsmith, D. Posterior leuko-encephalopathy syndrome as the presentation of renal artery stenosis. *J Renovasc Dis.* 2003; 2: 48–53