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### Imaging of Postpartum Encephalopathy: A Pictorial Essay

Authors

# Dr Shashibala J Yadav<sup>1</sup>, Dr Abhishek Jain<sup>2</sup>, Dr Rajesh Mahavir Shanklesha<sup>3</sup>, Dr Nikhil Ihare<sup>4</sup>

<sup>1</sup>Fellow in Diagnostic Neuroradiology, <sup>2</sup>Resident, <sup>3</sup>Fellow in Body Imaging, T.N.M.C & B.Y.L.Nair Charitable Hospital Mumbai,

<sup>4</sup>Consultant Radiologist IQRAA International Hospital & Research Centre Calicut, Kerala Corresponding Author

Dr Shashibala J Yadav

Fellow in Diagnostic Neuroradiology, T.N.M.C & B.Y.L.Nair Charitable Hospital Mumbai

### Abstract

Cerebrovascular events are relatively rare in young patients. They may be seen in some patients at a younger age if there is history of predisposing medical conditions like sickle cell disease, protein c, protein s or antithrombin deficiency. The incident of stroke in non-pregnant females of reproductive age group is reported to be around 10 per 100000 women-years. There is 3 fold rise in the risk of cerebrovascular accidents in pregnant women of same age group. The common causes of such events in young women include, oral contraceptive pills use, Systemic lupus erythromatosus (SLE), antiphospholipid antibody syndrome (APLAS), reversible cerebral vasoconstriction syndrome (RCVS), vasculitis syndromes like takayasu's arteritis, moyamoya disease and susac's syndrome. Adverse cerebrovascular events are more common in pregnancy, postpartum and puerperal period as compared to non-pregnant women of same age. The predisposing factors for postpartum acute cerebrovascular disease include pre-eclampsia, eclampsia, pre-existing chronic kidney disease, diabetes mellitus, congenital or acquired hypercolagulable states and puerperal sepsis. Some of the common cerebrovascular events seen in postpartum period include cerebral venous thrombosis, reversible cerebral vasoconstriction syndrome, posterior reversible encephalopathy syndrome and postpartum angiopathy. The diagnosis of these conditions is usually by neuroimaging. This paper is a pictorial essay on the neuroimaging of some of the common cerebrovascular events seen in postpartum period. Familiarity with these neuroimaging findings is important to differentiate these conditions from other causes of altered sensorium in postpartum period like postpartum psychoses, electrolyte imbalance and eclampsia.

**Keywords:** Postpartum encephalopathy, stroke, Magnetic resonance imaging, MR Angiography.

#### Introduction

Acute neurological conditions requiring critical care is uncommon in women of childbearing age unless they have conditions predisposing them for developing neurological problems such as Systemic lupus erythromatosus (SLE), antiphospholipid antibody syndrome (APLAS), protein c,

and Protein-S and antithrombin deficiency <sup>[1]</sup>. Young woman even without any of these condition are prone to develop acute neurological condition during pregnancy and postpartum period. One of the important and common causes of altered sensorium and seizures encountered in routine obstetric practice is eclampsia.

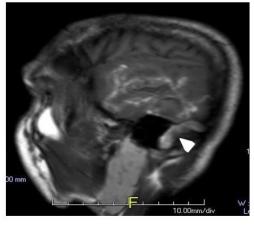
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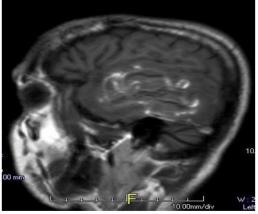
Nonetheless there are other conditions which are indirectly related to pregnancy and can cause post partum encephalopathy [2]. The predisposing factors for postpartum acute cerebrovascular disease include pre-eclampsia, eclampsia, preexisting chronic kidney disease, diabetes mellitus, congenital or acquired hypercolagulable states and puerperal sepsis [3]. Some of the common cerebrovascular events seen in postpartum period include cerebral venous thrombosis, reversible cerebral vasoconstriction syndrome, posterior encephalopathy syndrome reversible and postpartum angiopathy. These disorders carry considerable morbidity and mortality unless recognized and treated early. It's important to differentiate post partum psychoses electrolyte imbalance, eclmpsia and postpartum psychoses because of obvious implication such a diagnosis will have [4].

This pictorial essay focuses mainly on imaging findings of post partum encephalopathy with an emphasis on its early diagnosis. Such an early diagnosis will definitely have a positive impact on outcome of patients having such acute postpartum neurological condition.

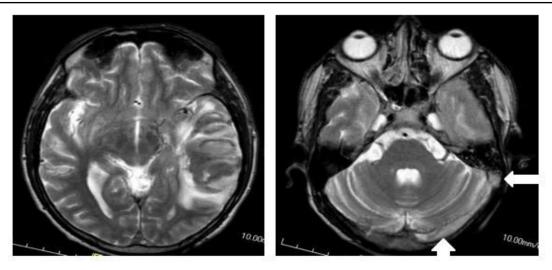
### **Pictorial Review:**

**Case 1**: Postpartum Cerebral Sinus Thrombosis A 29 years old female, 2<sup>nd</sup> gravida was admitted 10 days after LSCS with a history of severe headache since 6 hours. LSCS was uneventful, patient was apparently alright for a week post delivery when she developed above complaints and was advice for MR brain imaging. Headache was felt predominantly in occipital region. There was also a history of altered sensorium, nausea and vomiting. There was no history of seizures. On examination air entry was bilaterally equal. There were mild wheeze present bilaterally. Blood pressure was 110/70 and pulse was regular 82/min. Auscultation of cardiovascular system didn't reveal any abnormality. Pupils were bilaterally equal and reacting to light. There was no e/o any rash or any localizing sign of infection. There was no neck rigidity. Patient was admitted and IV fluids and antibiotics were started. Patient was kept nill by mouth and was sent for neuroimaging. MRI was done which showed left transverse sinus thrombosis causing hemorrhagic infarct in temporal lobe. On T1 weighted sagittal images it was seen as mixed signal intensity area predominantly hyperintense in left temporal lobe. Loss of flow void was noted in left transverse sinus (Figure 1). On T2 weighted images there was showing haematoma in left temporal lobe which is predominantly hypointense with adjacent perilesional edema (Figure 2). Further imaging by fast field echo images reveals blooming area in left temporal lobe consistent with hemorrhage. This blooming effect was also present in the area of left transverse sinus s/o thrombosis (Figure 3, 4). Finally MR angiography could conclusively demonstrate the thrombosis of left transverse sinus (Figure 5).

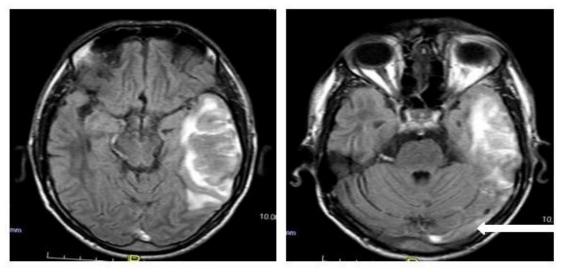




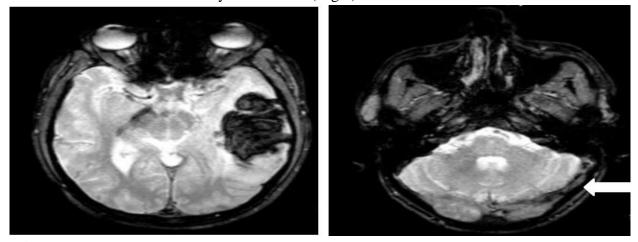
**Figure 1:** MRI brain showing left transverse sinus thrombosis leading to temporal lobe hemorrhagic infarct. T1 weighted sagittal images reveals mixed signal intensity predominatly hyperintense signal in left temporal lobe. Also there is loss of flow void in left transverse sinus (white arrowhead).



**Figure 2:** T2W axial images of same patient showing hematoma in left temporal lobe which is predominantly hypointense with adjacent perilesional edema (Left). White arrow pointing towards loss of flow void in left transverse sinus extending up to sigmoid sinus (Right).



**Figure 3 :** T2W FLAIR axial images reveals left temporal hemorrhage with perilesional edema and loss of flow void in transverse sinus marked by white arrow (Right).



**Figure 4:** FFE axial images reveals blooming area in left temporal lobe consistent with hemorrhage (Left). Also left transverse sinus shows blooming foci which is consistent with finding of transverse sinus thrombosis (Right).





Figure 5: MR angiography reformatted images showing left transverse sinus thrombosis

# Case2:Reversible Cerebral Vasoconstriction Syndrome

35 years old female, one week post normal delivery presented with thunderclap headache which was diffuse in nature associated with nausea, vomiting. There was no history of any medical illness in past. On examination her blood pressure was normal. MR brain was advice for further evaluation which showed altered signal intensity in right frontal lobe which was hypointense on T1 and hyperintense on T2 (Figure

6). Axial images on T2 FLAIR showed hyperintense signal in right frontal lobe (Figure 7). DWI images revealed gyriform pattern hyperintensity in right frontal lobe with signal drop on ADC suggestive of restricted diffusion (Figure 8). Finally MR angiography showed gross attenuation of luminal caliber of bilateral middle cerebral artery these narrowing was predominantly seen on right side (Figure 9). Findings were demonstrated on reformatted axial and coronal images (Fig. 10).

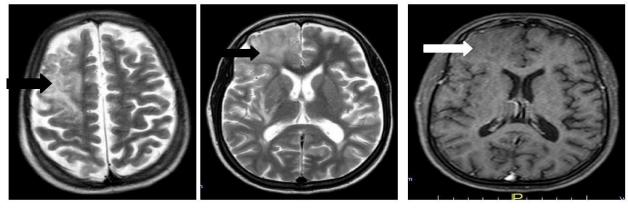


Figure 6: Altered signal intensity area hyperintense on T2 and hypointense on T1 in right frontal region

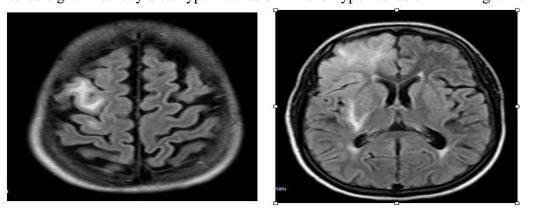
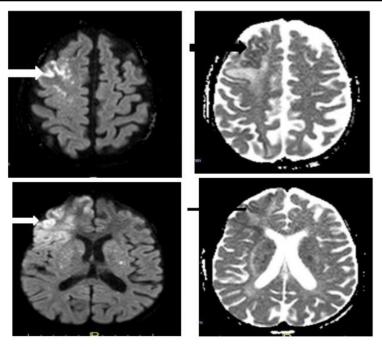
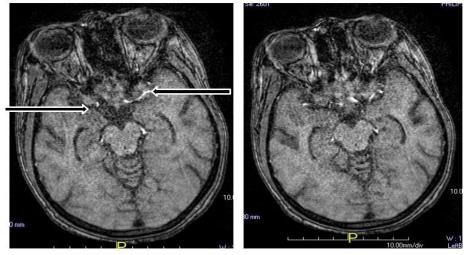


Figure 7: T2 FLAIR axial images reveals hyperintense signal in right frontal lobe



**Figure 8:** DWI reveals gyriform pattern hyperintensity in right frontal lobe (white arrow) which show signal drop on ADC suggestive of restricted diffusion (black arrow)



**Figure 9:** MR angiography source images reveal gross attenuation of luminal caliber of bilateral middle cerebral artery (right>left)



**Figure 10:** MR Angiography reformatted axial and coronal images reveals gross attenuation of luminal caliber of bilateral middle cerebral artery (right>left)

## Case 3: Posterior Reversible Encephalopathy Syndrome.

A 31 years old female presented with severe occipital headache, nausea, vomiting and two episodes of seizure and photophobia. There was a history of having delivered a full term male by LSCS 10 days back. The indication of LSCS was oligohydramnios. Immediate post LSCS period was uneventful. But since 2 days she had a history of nausea vomiting and headache. Today she got 2 episodes of generalized tonic clonic seizures. Each episode lasted for about 2-3 minutes followed by

loss of consciousness for 10-15 minutes. Patient remained drowsy in between the episodes of seizure and the time of admission in hospital also the patient had altered sensorium. MRI was done which showed altered signal intensity areas in lobes bilateral parietal which appeared hyperintense on T2 weighted images and on T2 FLAIR (Figure 11). T2 FLAIR images of same patient showing hyperintense signal in bilateral parietal lobe MR Angiography reformatted, DWI and ADC images were unremarkable (Figure 12,13,14).

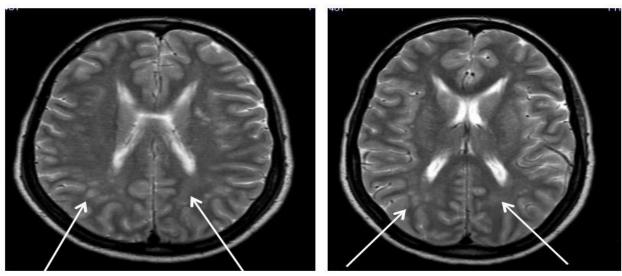
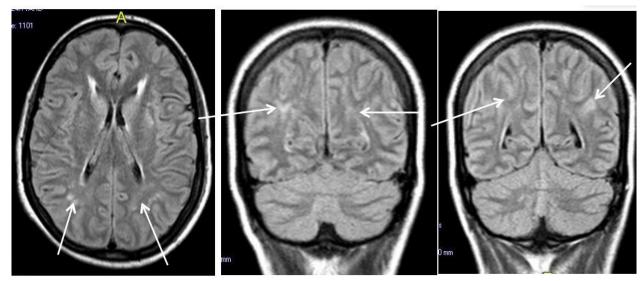


Figure 11: T2WI reveals hyperintense signal in bilateral parietal lobe (white arrow).



**Figure 12:** T2 FLAIR images of same patient showing hyperintense signal in bilateral parietal lobe (white arrows).

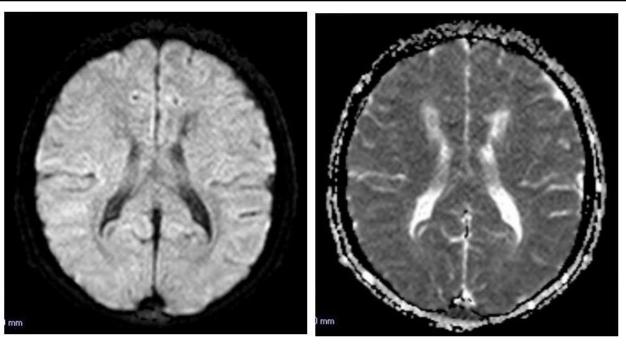
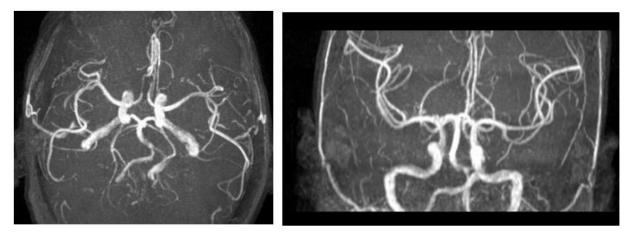


Figure 13: DWI and ADC images were unremarkable

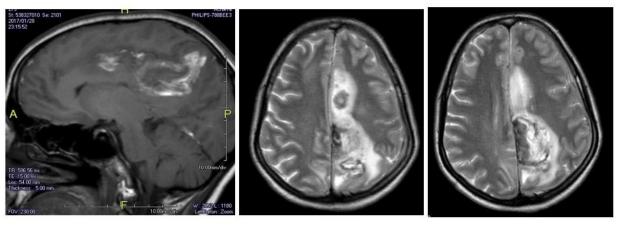


**Figure 14:** MR Angiography reformatted images were normal.

### **Case 4: Postpartum Cerebral Angiopathy**

A 28 years old female presented with sudden onset of severe headache, one episode of seizure and altered sensorium 8 days after normal delivery. Convlusions were focal involving right side of the body. Patient had loss of consciousness after convulsions which lasted for 10-15 minutes. Also there was history of post-ictal confusion. There was no significant history of any neurological condition in past. Also there was no past history of diabetes, hypertension or any other major illness. Antenatal, natal and immediate post

natal period was uneventful. There was no history of eclampsia. In view of altered sensorium and h/o seizures neuroimaging was advised. An MRI was done which showed mixed signal intensity areas predominantly hypointense in left parietal lobe parafalcine region. Corresponding T1W sagittal images showed hyperintense signal surrounded by perilesional edema. T2 FLAIR and FFE axial images confirmed the presence of hematoma and MR Angiography reformatted coronal image were unremarkable (Figure 15-18).



**Figure 15 :** T2W axial images reveals hematoma in left parietal lobe parafalcine region showing mixed signal intensity predominantly hypointense, corresponding T1W sagittal image show hyperintense signal surronded by perilesional edema.

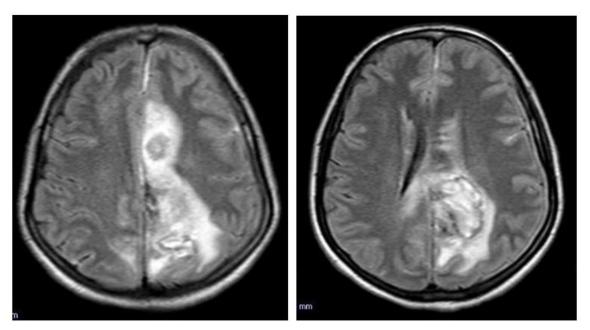


Figure 16: T2 FLAIR images of same patient showing hematoma.

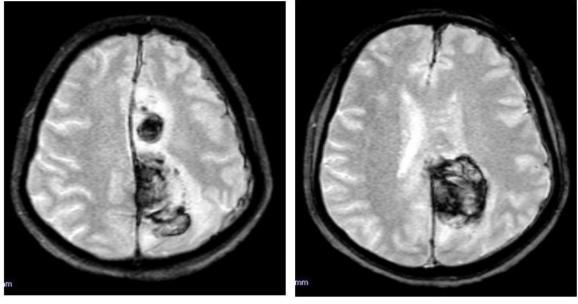


Figure 17: FFE axial images showing hypointense signal supporting the diagnosis of hematoma.



**Figure 18:** MR Angiography reformatted coronal image is unremarkable.

### **Discussion**

Acute neurological conditions in post partum period are usually related to eclampsia. Other conditions which may be the cause of neurological manifestations like seizures, focal deficit and altered sensorium are dural sinus thrombosis, cerebral vasoconstriction syndrome, posterior encephalopathy syndrome reversible postpartum angiopathy. Eclampsia is usually well recognized by obstetricians and usually there is history of hypertension and albuminuria during pregnancy. Other causes of acute neurological manifestations like dural sinus thrombosis, stroke or angiopathy should be diagnosed in early phase and treated promptly as any can lead to devastating complications [5]. Since all these conditions may initially present as headache there is usually a history of initial treatment by oral anaelgesics until serious neurological features develop. Due to non-specific symptoms in initial stages neuroimaging is of utmost importance for early diagnosis <sup>[6]</sup>.

Dural sinus thrombosis may manifest as hemorrhagic or ischemic complications. It is more common in females and it is of utmost importance that obstetrician and those involved in care of pregnant women should know that postpartum period is associated with a significant increase in incidence of cerebral venous thrombosis. Many factors predispose women for development of

sinus thrombosis including hypercolagulable state seen in pregnancy, infection, and antiphospholipid antibody syndrome and prothrombin gene mutation. Patient usually present with severe headache, convulsions, and papilledema if severe enough to cause raised intracranial pressure. Though CT scan can be done MRI and MR venography are the modalities of choice for the diagnosis of dural sinus thrombosis <sup>[7]</sup>.

Reversible vasoconstriction syndrome is another cause of headache and neurological manifestation in postpartum period. Patient usually present with acute-onset severe (thunderclap) headaches which may be accompanied by convulsions, features simiar to or suggestive of stroke, The encephalopathy. condition is reversible and prognosis is good. However, some patients may experience severe spasm and massive infarct. Rarely may it prove to be fatal. The hallmark finding is the finding of reversible vasoconstriction of the cerebral vasculature mainly arteries. Convulsions caused by reversible vasoconstriction syndrome should be differentiated from seizures caused by eclampsia, dural sinus thrombosis and angiopathy. The diagnosis is usually by CT, MRI and MR angiography. MR angiography may show attenuation of luminal caliber of cerebral arteries [8].

Posterior reversible encephalopathy syndrome (PRES) is characterized by headache, altered sensorium, seizures, and visual disturbances. Additionally, there are characteristic imaging features associated with the syndrome which often include focal regions of symmetric edema in the parenchyma of posterior portion of brain. As the name suggests It is usually reversible but some patient may develop complications leading to stroke and variable intensity of neurological deficits. MRI may show altered signal intensity areas in bilateral parietal lobes [9].

Postpartum angiopathy (PPA) is a vasoconstriction syndrome of uncertain etiology that affects medium and large sized cerebral arteries. Postpartum angiopathy may cause ischemic stroke. 2/3 of the cases of postpartum angiopathy present within 1 week after delivery.

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The diagnosis of postpartum angiopathy is made angiography. It may show segmental narrowing and dilatation in large and mediumsized cerebral vessels .MRI may show areas of T2/FLAIR hyperintensities at any location, especially in watershed areas of brain. It is generally self limiting and the signs and symptoms usually subside in 1- 3 months but some patients may have residual neurological manifestations for longer period of time. It is important to know that angiography may be normal in postpartum angiopathy in initial days and if there is strong suspicion of angiopathy repeat angiography should be done a few days later [10].

### **Conclusion**

Cerebrovascular events in postpartum period are relatively common. These events may initially present with non-specific symptoms like headache, giddiness and vomiting. The familiarity with the neuroimaging features of postpartum encephalopathy may help in early diagnosis and prompt treatment thereby preventing further complications.

### Conflict of interest: None

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