Original Research Article

Acute neurological disorders in women during pregnancy and peurperium

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Abstract

Neurological disorders in women during pregnancy and puerperium are a significant cause of morbidity and mortality in pregnancy. There are certain neurological conditions which are specifically related to physiological changes during this period (eg: eclampsia, Sheehan's syndrome, posterior reversible encephalopathy syndrome) and some disorders which have increased risk but not specific to women in pregnancy and puerperium (eg: cerebral venous thrombosis, infarction). As radiologists, an understanding of the patho physiological mechanism and imaging findings associated with these various conditions is necessary in timely diagnosis and initiating therapy which in turn helps prevent complications to both them other and the fetus. Any prophylaxis against these events should be particularly targeted to postpartum women, indicating the need to better identify pregnant women at increased risk.

Key Word: Neurological disorder, Cerebral venous thrombosis, Posterior Reversible Encephalopathy syndrome, pregnancy and puerperium.

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INTRODUCTION

Pregnancy and the postpartum period involves many physiological and anatomical changes which makes them vulnerable for many neurological conditions, both-exacerbation of pre-existing illness and development of acute neurological disorders. Headache and seizure remains the commonest symptoms of neurological disorders in this period. Therefore the clinicians should be sensitised to the "red flag signs"(head ache and seizures) which helps in initiating prompt diagnostic investigations. As radiologists we should be familiar with these cerebrovascular conditions encountered in these young which is very important for initiating early therapy and preventing complications to both the mother and the fetus. Evaluation of the women in puerperal period usually starts with unenhanced CT. However normal CT findings cannot reliably exclude many of the acute neurological disorders in pregnancy and puerperium, which makes MRI the modality of choice with its ability to detect subtle white matter changes and assess the intracranial vasculature[1]. In pregnant women despite of ALARA (As Low As Reasonably Achievable) Principles allowing CT to be used safely in evaluating the mother's head and neck first line investigation is usually MRI(2). In this article we emphasize on the acute neurologic disorders that occur during the course of pregnancy and the postpartum period.

Learning Objectives

- To list the various acute neurological disorders in women of pregnancy and puerperium.

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- To identify the principle imaging features of theses variable disorders.
- To describe the pathophysiological correlation between the common entities and correlate with imaging findings.

Cerebrovascular complication

Acute neurological complications in the pregnancy and postpartum can be classified for the better understanding of the disorders. (Fig1)

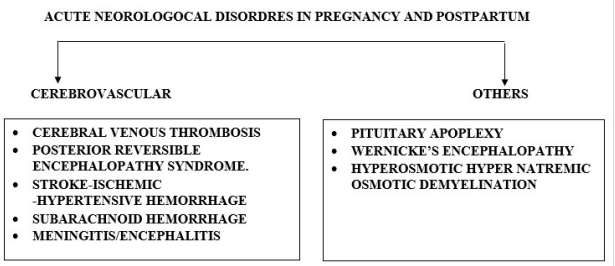

Figure 1: Types of Acute neurological disorders in pregnancy and uerperium.

Cerebrovascular complications

Cerebral Venous Thrombosis (CVT)

Cerebral venous thrombosis refers to thrombosis on intracranial venous channels including dural venous thrombosis, cortical venous thrombosis and deep vein thrombosis. CVT may occur anytime during the course of pregnancy and the puerperium, but the risk is highest during the first 2 weeks of the puerperium[3]. CVT accounts for 6% of maternal deaths and highest being for young mothers and after caesarean section[4]. The clinical presentation varies depending on the severity and extent of thrombosis as well as the mode of onset. It may vary from headache to coma. In sepsis cavernous and lateral sinus thrombosis are mostly involved whereas superior sagittal sinus is most commonly involved in non-septic CVT[5].

Pathophysiology

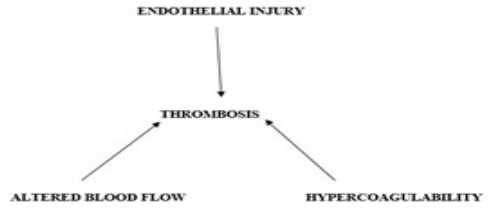

Figure 2: Diagram showing Virchow's triad.

Pregnancy induces several changes in coagulation system rendering it a prothrombotic state. (Fig 2) Hypercoagulability worsens further after delivery as a result of volume depletion and trauma. Stagnant flow contributes few percent due to episode of dehydration,dural puncture,and sometimes with hyperosmolar contrast agent.During puerperium additional risk factors include infection and instrumental delivery or Caesarean section[5].

Imaging features:

CT

Hyper-attenuation of the thrombosed sinuses with or without venous infarction (Figure: 3) .CT venography shows focal venous cut-offs or filling defects in the sinus, with peripheral enhancement-that is, the "empty-delta" sign[6] However, it may take 7–10 days for the empty delta sign to show on CT scans after the onset of symptoms .MR imaging is more sensitive than CT in early detection of thrombosis and more accurate in depicting the extent and complications of CVT[6]

MRI

In T1, T2 and FLAIR sequences it shows high signal intensity of the dural venous sinuses an in SWI it shows blooming / loss of normal flow void in adjacent dural sinus. MR venography reveals non visualisation of thrombosed sinus[6].Figure:4, 5, 6. Parenchymal signs of CVT include diffuse mass effect, localized sulcal effacement, parenchymal infarcts /edema. Venous infarcts typically do not conform to the arterial territories and are often associated with hemorrhage at the gray-white matter interface.

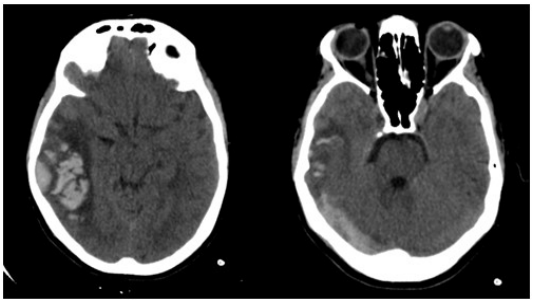

Figure 3: 25 year old female at postpartum day 5 c/o 1 episode of seizure Non contrast CT revealed atypical hemorrhage in right temporal lobe. Thrombosed right transverse sinus is seen.

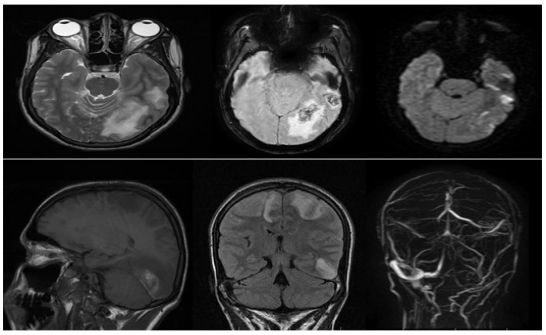

Figure 4: 24 year old lady with h/o head ache on post-partum 17th day, Axial T2, SWAN, DWI, Sag T1, Coronal T2 FLAIR and MR Venography reveals haemorrhagic venous infarct with perilesional edema is seen in the left temporal lobe secondary to CVT. MRV

shows intrinsic filling defects in superior sagittal sinus, left transverse, sigmoid sinus, IJV and superficial cortical veins.

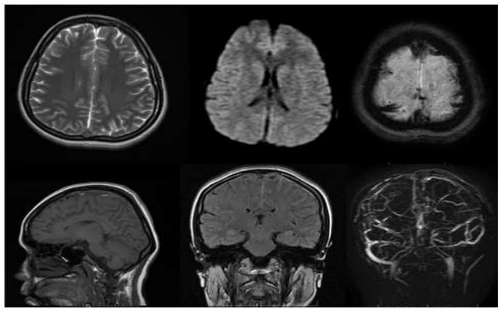

Figure 5: 20 y old lady with h/o head ache on post partum day 5.Non visualisation of superior sagittal, inferior sagittal, straight sinus, both transverse and left sigmoid sinuses on MRV with corresponding high signal on T1 images - in keeping with cortical venous thrombosis. On SWAN images, there is suggestion of blooming involving high parasagittal veins and superior sagittal sinus.

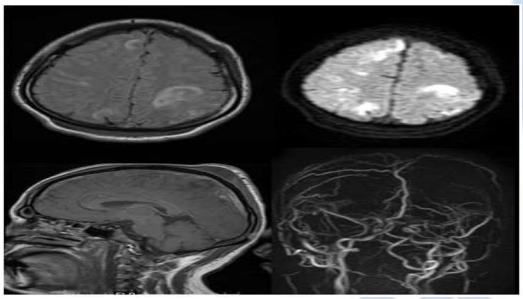

Figure 6: 23 y old women with 5 months of amenorrhea day, c/o headache, Axial T2 FLAIR, DWI, Sag T1 and MRV reveals venous infarct in bilateral frontal lobe secondary to thrombosis of superior sagittal and bilateral transverse sinuses.

Posterior reversible encephalopathy syndrome (PRES)–Eclamptic Encephalopathy:

Eclampsia is defined as new onset of seizures and/or coma during the pregnancy, labour or peurperium in the setting of preeclampsia. PRES is one of the most common complication of preeclampsia, eclampsia, and pregnancy-induced severe hypertension. The term PRES can be a misnomer as the syndrome can involve anterior circulation territories as well[7]. Patients with PRESS presents with headache, generalized seizures, altered mentation, visual disturbance and severe hypertension.

Pathophysiology

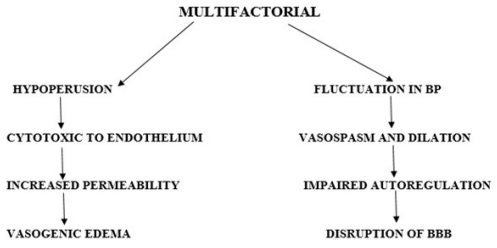

Figure 7: Diagram illustrating pathophysiology of PRES

The predilection for the posterior circulation and watershed zones is believed to be related to its sparse vasomotor sympathetic innervations (Figure: 7).[8]

Imaging findings in PRESS

On CT imaging bilateral symmetrical areas of hypodensity, predominantly involving parieto-occipital lobes are seen[9].Figure:8

MR

Symmetric edema in the parietal and occipital lobes at the grey matter-white matter junctions. Similar changes may also beseen in the frontal lobes, inferior temporo-occipital junction, and cerebellum[9].Figure 9, 10 T1-weighted images shows low signal intensity. T2- weighted/FLAIR images shows high signal intensity in the posterior cortex and subcortical white matter. DWI shows no diffusion restriction, useful in distinguishing the reversible vasogenic edema from the cytotoxic edema of complete infarction.

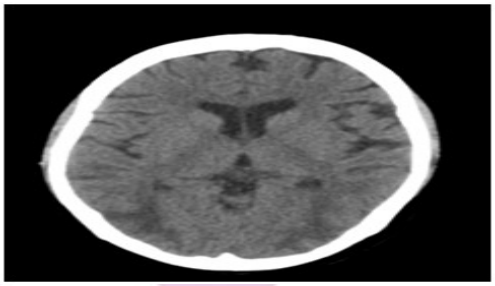

Figure 8:21 year old female with h/o postpartum eclampsia Non contrast CT reveals symmetrical areas of hypodensity noted involving bilateral parieto-occipital lobes-Suggestive of PRES.

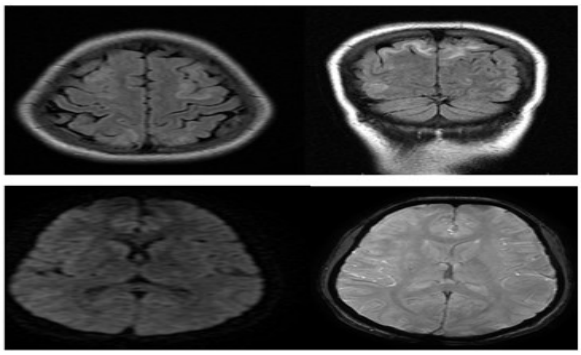

Figure 9: 30 years old female with h/o seizures on post-partum 7th day. Axial/Coronal T2 FLAIR,DWI and SWAN reveals symmetrical FLAIR hyperintense signals involving cortical and subcortical areas in the bilateral parietal region with no restricted diffusion-Suggestive of PRES

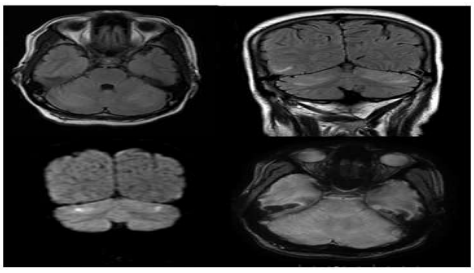

Figure 10:20 year old postpartum female with antepartum pre-eclampsia now co seizure Axial and Coronal T2 FLAIR,DWI and SWAN reveals high signal intensity involving bilateral cerebellar hemisphere with diffusion restriction-Indicative of PRES.

Stroke

Women in pregnancy and puerperium are in a hyper coagulate state and hence are prone for both ischemic and hemorrhagic stroke. Strokes, both ischemic and hemorrhagic are major contributors to morbidity and mortality during pregnancy and the puerperium. The overall incidence of ischemic stroke during pregnancy and postpartum is low 3.5-5 per 100 000 pregnancies in the developed world (10).But it has to be considered that when compared to stroke in the young, as a broader group those related to pregnancy accounted for 12% to 35% of events in this otherwise low-risk population[11]

Pathophysiology of stroke:

Factors leading to hypercoagulability of blood include increased levels of fibrinogen, factor VII, factor VIII, and factor X; low levels of inhibitors of the coagulant protein S; elevated levels of inhibitors of protein C;and an enhanced ability to neutralize heparin[11]

Hemorrhage is mostly associated with preeclampsia / eclampsia. Physiological changes of pregnancy including increased blood volume, rising blood pressure, and changes in vascular tone contribute to hemorrhage.

The clinical manifestations like stroke in any other population depends on the site of insult and will result in symptoms ranging from dense hemiplegia to hypoesthesia.

Imaging features in stroke

Table 1: Ischemic stroke

	NCCT	TI	T2/Flair	DWI	Post Gadolinium
Hyperacute	--------	Normal	Normal Flair Positive by 3hrs	Restriction with low ADC	Lack of Flow on MRA
Acute	Hypodense Mass wffect	Hypo	Hyper	Restriction with low ADC	Meningeal

Table 2: Hemorrhagic stroke

	Haemoglobin	CT	T1	T2	GRE
Hyperacute	Oxy Hb	Bright	Iso to Slightly hyperintense	Hypointense	Hyperintense than grey matter with hypointense rim
Acute	Deoxy Hb	Increasingly bright	Iso to hypointense	Hypointense periphery which extends inwards	Hypointensity Blooming

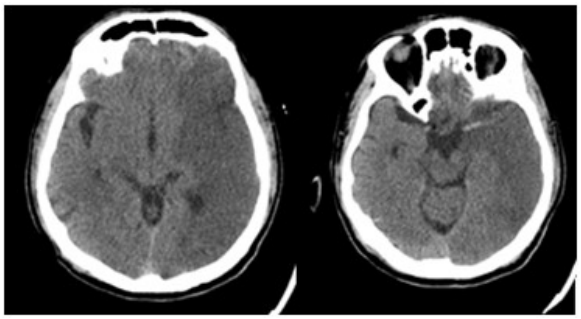

Figure 11:33 year old postpartum women with dense right hemiplegia, non contrast CT shows hypodense are in left fronto-parieto-temporal lobe with left dense MCA sign-Suggestive of left MCA infarct.

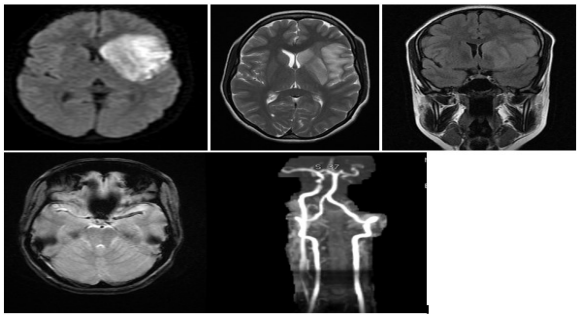

Figure 12: 25 year old woman with 9 months of amenorrhea with right sided weakness, Axial DWI,T2,Coronal T2 FLAIR,SWAN and MRA shows left MCA territory infarct secondary to thrombosis involving left ICA and MCA.

Subarachnoid hemorrhage (SAH)

Pregnancy may increase the risk of aneurysm rupture due to the hemodynamic and hormonal alterations and contributes to commonest cause of SAH in pregnancy. SAH due to aneurysmal rupture commonly occurs in young Primigravida during the third trimester Primary nonaneurysmal SAH due to pregnancy- induced hypertension is an extremely rare event.[12]

Pathophysiology of SAH

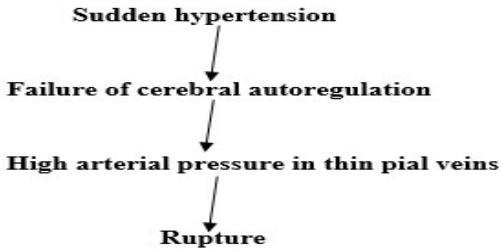

Figure 13: Illustration demonstrating pathophysiology of SAH.

Imaging findings in SAH

CT: Hyper-attenuation along the subarachnoid space.(Figure:14)

MRI:

FLAIR: hyperintensity in the subarachnoid space.

SWAN: Blooming along cortical sulci and subarachnoid space.

MR angiography and MR venography: Helps in detecting causative aneurysm or another source of bleeding. Figure:15.

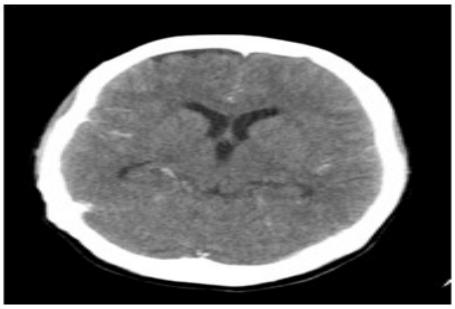

Figure 14: 25 year old woman at term c/o sudden onset of headache with decreased responsiveness, non contrast CT reveals SAH along bilateral fronto-parieto-temporal cortical sulci.

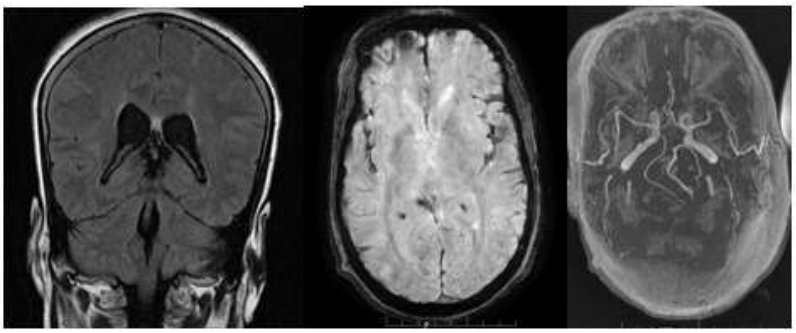

Figure 15: 20 year old female postpartum c/o severe headache Coronal FLAIR hyperintensities along cortical sulci, blooming along bilateral sylvian fissure and cortical sulci. MRA did not reveal any aneurysm.

Pituitary apoplexy

Pituitary apoplexy is acute hemorrhagic infarction in an existing pituitary adenoma or otherwise physiologically enlarging pituitary gland as in pregnancy. Clinically, the patient may present withsevere headache, vomiting, and visual disturbances including visual field defects and restricted eye movements. Patients may also develop dizziness or altered mental status, thought to be the result of hemodynamic instability presumably due to acute hypopituitarism .Pituitary apoplexy's a rare occurrence during pregnancy and may be thought of as a presentation of pituitary micro adenoma.

Imaging findings in pituitary apoplexy

CT and MR imaging reveals hemorrhage in a prominent pituitary gland.Figure:15

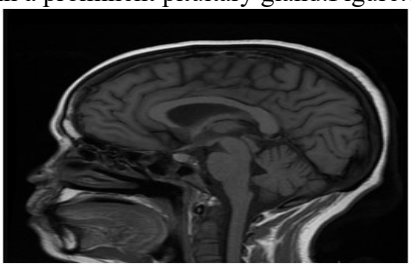

Figure 15: Sagittal T1W image reveals hyperintensity at pituitary-Pituitary apoplexy.

Wernicke's Encephalopathy

It is acute severe neuropsychiatric syndrome which results from thiamine deficiency. It is usually seen in alcoholics but can be seen in any malnourished state, especially in pregnancy with hyperemesis. The prevalence in non-alcoholic patients developing Wernicke's encephalopathy varies from 0.04% to 0.13% (13) It is characterised by the classic triad of encephalopathy, ophthalmoplegia, and/or nystagmus and ataxia.

Pathophysiology of Wernicke's encephalopathy.

Thiamine is an important co-enzyme in several biochemical pathways in brain, mainly involved in glucose metabolism and cerebral energy utilisation. Decreased alpha-ketoglutarate dehydrogenase activity results in cytotoxic edema (14).

MR imaging features in Wernicke's encephalopathy

T2/FLAIR: Symmetrical hyperintensities in thedorsomedial thalami, mammillary bodies, tectal plate, and periaqueductal area. DWI:May or may not show diffusion restriction.Figure:16

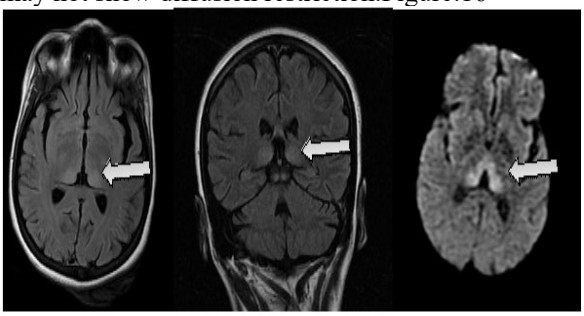

Figure 16:20year old patient at 5months of amenorrhea with altered sensorium Axial/Coronal T2 FLAIR Axial DWI reveals abnormal symmetrical hyperintensity at bilateral thalami and mammillary bodies-Wernick's encephalopathy.

Definite diagnosis of Wernicke's encephalopathy is based on the clinical manifestations and rapid reversal of symptoms with thiamine. Determination of blood transketolase activity and thiamine pyrophosphate reflects the thiamine status in the body[15].

Postpartum hypernatremia and osmotic demyelination

Hypernatremia can cause osmotic demyelination, encephalopathy and rhabdomyolysis. Neurological complaints secondary to extrapontinemyelinolysis (EPM) and rhabdomyolysis caused by hypernatremia are infrequent.

Pathophysiology

Restriction of water intake for females in post-partum periodmay exacerbate dehydration and which in turn leads into hypernatremia. Hypernatremia leads into osmotic demyelination[16]

MR imaging features of hypernatremia osmotic demyelination.

T2 and FLAIR: Hyperintensities in pons and extrapontine sites including basal ganglia, thalami, and cerebral white matter. Symmetric trident-shaped hyperintensity/Wineglass appearance in the central pons is a characteristic finding with sparing of ventrolateral pons and the pontine portion of corticospinal tracts[17].

Atypical sites of extra-pontine myelinolysis include grey matter, white matter, corpus callosum, splenium, cerebellum, hippocampus, and external capsule. Other rare sites include midbrain, subthalamic nuclei, claustrum, hypothalamus, medulla, and amygdala.

DWI: Shows diffusion restriction.

CONCLUSION

The dynamic changes occurring during pregnancy and puerperium may induce a variety of non-obstetric complications in central nervous system. It is important for neurologists to make the distinction between the benign and more serious neurologic signs, symptoms, and complications seen in pregnant and puerperial period and thus initiating timely imaging studies. Radiologists should be sensitised to the pathophysiological and imaging features of these disorders which is the key factor in more precise and early diagnosis, ensued by an early treatment by the clinicians.

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