Original Research Article

The challenge of neuroimaging during pregnancy

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INTRODUCTION

Central nervous system conditions may affect every aspect of female reproduction from fertility to lactation. Various neurologic disorders can occur during pregnancy, where they can be a direct cause of pregnancy or can be exacerbated by pregnancy. It is noteworthy that some disorders can manifest for the first time during pregnancy.¹ The diagnosis and management of the neurologic disorders can be challenging during pregnancy for a myriad of reasons and often require a stepwise, multidisciplinary approach.² Diagnosis of neurologic disorders often requires the use of magnetic resonance imaging (MRI) and computed tomography (CT).

Although avoidance of ionizing radiation is strongly recommended particularly during the first trimester due to its negative effects on the fetus, it sometimes needs to be used during imaging.¹,² Some neurologic diseases have little or no effect on pregnancy, while others can increase the risk of adverse pregnancy outcomes.³ Pregnancy-related complications of elevated blood pressure and fluid retention, such as pre-eclampsia and more seriously eclampsia, may also cause neurological problems.³ Cerebrovascular diseases that occur during pregnancy can be divided into two broad categories: thrombosis/ischemia, including arterial and venous infarction, and hemorrhage, including intracerebral and subarachnoid hemorrhage.⁴ Regarding thromboembolic...
disease, it is well known that the normal physiologic changes of pregnancy predispose women to develop neurological disorders, including strokes during pregnancy and the puerperium. Maternal physiological alterations occur during pregnancy as a consequence of the variations of the hormonal status, involving the hemostatic and hemodynamic systems.8 Emergent evaluation of headache, seizure and coma of patients during pregnancy and just after delivery requires rational selection of acute neuroimaging studies, yet guidelines do not exist. The objective of this study was to examine the performance of magnetic resonance imaging and computed tomography in the diagnosis of maternal neurologic disorders and to examine the outcome of pregnancies complicated by abnormal neurologic imaging.

**METHODS**

This was a multi-center retrospective observational study of clinically suspected cases of neurological disorders during pregnancy that demonstrated abnormal findings on imaging. The study was conducted following approval of the research ethics committee at the three centers that participated in the study. Study period was (January 2006 to January 2016).

**Inclusion criteria**

- Patients in the second or third trimester or postpartum, and patients with generalized seizures, high blood pressure (140/90 mmHg), and proteinuria.

**Exclusion criteria**

- Patients with neurological disorders not directly related to pregnancy as well as claustrophobic patients. Initial MRI or CT was performed within 24 hours of the onset of symptoms.

**RESULTS**

The selected patients were evaluated on the basis of detailed history, clinical and laboratory examination. CT scans and MRI were strictly limited to the area of interest. A total of 20 pregnant women with neurological disorders (13 antepartum and 7 postpartum) were studied. The cases included eclamptic encephalopathy, posterior reversible encephalopathy syndrome (PRES), and subarachnoid hemorrhage (SAH). The majority of patients delivered by C-section. Demographic data, neurological disorders as well as CT and MRI findings of the 20 patients are shown in Table 1.

| Patient no. | Age and time of pregnancy | Parity, number of pregnancy, mode of delivery | Blood pressure (mmHg), proteinuria | Neurologic symptoms | Location of lesions at initial CT, MRI | Residual neurologic deficits | Follow-up neuroimaging findings and baby outcome |
|-------------|---------------------------|---------------------------------------------|-----------------------------------|---------------------|-----------------------------------|-----------------------------|-----------------------------------------------|
| 1           | 22 years 37 weeks         | G1P0A0 NVD                                 | 160/110 (Eclampsia before delivery) | Convulsion          | CT only; Occipital hemorrhage     | No                          | Normal alive baby                          |
| 2           | 29 years 30 weeks         | G1P0A0 C/S                                 | 190/110 (Eclampsia before delivery) | Headache, vomiting, blurry vision, seizure | Occipital parietal edema | No                          | Normal ICN alive                           |
| 3           | 19 years 24 weeks         | G3P0A2 C/S                                 | 150/75 (Eclampsia before delivery) | Headache, convulsion | SAH frontal Parietal edema cortex | No                          | Normal ICN dead                            |
| 4           | 39 years                  | G4P2A1 C/S                                 | 170/110                           | Disorientation, SAH diffuse | No                          | Normal                                   |
| Patient no. | Age and time of pregnancy | Parity, number of pregnancy and mode of delivery | Blood pressure (mmHg), proteinuria | Neurologic symptoms | Location of lesions at initial CT, MRI | Residual neurologic deficits | Follow-up neuroimaging findings and baby outcome |
|------------|---------------------------|-----------------------------------------------|----------------------------------|--------------------|--------------------------------------|-----------------------------|---------------------------------------------|
| 5          | 19 years 37 weeks         | G2P0A1 C/S                                    | 170/120 (Eclampsia before delivery) p+3 | Headache, vomiting, seizure | Cerebellum hemorrhagia | No                          | Normal Alive baby                          |
| 6          | 36 years 30 weeks         | G5P1A3 C/S                                    | 150/100 (Eclampsia before delivery) p+3 | Disorientation, Headache, vomiting, blurry vision | Parietal sagittal sinus hemorrhage paretial white gray matter interface | No                          | Normal Dead baby                           |
| 7          | 40 years 35 weeks         | G2P1A0 C/S                                    | 170/110 (Eclampsia before delivery) p+3 | Impaired vision, convulsion | Parietal SAH hemorrhage sulci cortex | No                          | Normal Alive baby                          |
| 8          | 32 years 36 weeks         | G2P2A0 C/S                                    | 150/90 (Eclampsia before delivery) p+3 | Impaired vision, convulsion | Occipital edema PRES | No                          | Normal Alive baby                          |
| 9          | 33 years 39 weeks         | G1P0A0 C/S                                    | 140/100 Preeclampsia p+3          | parastesie         | spinal cord Hematoma                   | No                          | Normal Dead baby                           |
| 10         | 25 years 34 weeks         | G1P0A0 C/S                                    | 150/90 Preeclampsia p+3          | Headache, vomiting | Parietal hemorrhage sagittal sinus | No                          | Refused                                    |
| 11         | 39 years 39 weeks         | G2P1A0 C/S                                    | 140/90 (Eclampsia before delivery) p+3 | Disorientation, seizure, decreased level of consciousness, opens eyes to painful stimuli, coma | Edema frontal pon cerebellum impression PRES | No                          | Not performed alive baby                   |
| 12         | 25 years 36 weeks         | G1P0A0 C/S                                    | 170/110 (Eclampsia before delivery) p+3 | Headache, convulsion | Occipital parietal edema corpus calloso | No                          | Normal Alive baby                          |
| 13         | 24 years 37 weeks         | G2P1A0 C/S                                    | 170/110 Preeclampsia p+3          | Headache, vomiting, blurry vision, seizure | Occipital talamus parietal edema | No                          | Normal Alive baby                          |
| 14         | 28 years 7d pp            | G3P3A0 C/S                                    | 150/75 Preeclampsia p+3           | Headache, weakness | SAH basales ganglia Parietal         | No                          | Normal Alive baby                          |
| 15         | 31 years 3d pp            | G3P3A0 NVD                                    | 170/110 Preeclampsia p+3          | Disorientation, Headache | SAH PRES brain edema parietal sulcus | No                          | Normal Alive baby                          |
| 16         | 26 years 7d pp            | G2P2A0 NVD                                    | 170/110 Preeclampsia             | headache, loss of consciousness | PRES occipital and temporal cortex | No                          | Normal Alive baby                          |
| 17         | 29 years 8 hours pp       | G4P0A3 C/S                                    | 150/100 Preeclampsia             | seizure            | Basal ganglia PRES                    | No                          | Normal Alive baby                          |
| 18         | 38 years 5d pp            | G2P1A0 NVD                                    | 150/90                           | headache           | Cerebellum hemorrhage                 | No                          | Normal Alive baby                          |
| 19         | 19 years 4d pp            | G2P1A1 C/S                                    | 150/90                           | weakness           | Frontal parietal edema PRES          | No                          | Normal Alive baby                          |
| 20         | 20 years 2d pp            | G1P1A0 C/S                                    | 170/110 (Eclampsia after delivery) | Headache, nausea, vomiting | Frontal parietal edema thalamus      | No                          | Normal Alive baby                          |

D pp: days postpartum, GPA: number of pregnancies, parity and abortion, C/S: cesarean section, NVD: normal vaginal delivery
Table 2: Distribution of age and type of eclampsia.

| Age of patient (years) | No. of patients | Percent |
|------------------------|-----------------|---------|
| 19-30                  | 12              | 60.0%   |
| 31-40                  | 8               | 40.0%   |

| Type of eclampsia       | No. of patients | Percent |
|-------------------------|-----------------|---------|
| Preeclampsia            | 8               | 40.0%   |
| Antepartum eclampsia    | 10              | 50.0%   |
| Postpartum eclampsia    | 1               | 5.0%    |
| None (didn’t have any of the above) | 1              | 5.0%    |

Most of the women (60%) presented with age between 19 to 30 years (12 cases) while 8 cases were between 31 and 40 years (Table 2). The mean age of the study population was 28.65±7.16 years. 8 patients (40.0%) presented with preeclampsia, 10 patients (50.0%) presented with antepartum eclampsia while one patient (5.0%) had postpartum eclampsia (Table 2). The mean age in women with preeclampsia, antepartum and postpartum eclampsia was comparable (26.88±4.39 versus 30.00±8.39 years versus 20 years respectively; p=0.272) (Table 3). 50% of patients with preeclampsia had headache compared to 40% of patients with antepartum eclampsia (p=0.494) (Table 3).

Table 3: Mean age and headache rate with respect to preeclampsia, antepartum eclampsia, postpartum eclampsia.

|                        | Preeclampsia (n=8) | Antepartum eclampsia (n=10) | Postpartum eclampsia (n=1) | P-value |
|------------------------|--------------------|-----------------------------|---------------------------|---------|
| Age (years)            | 26.88±4.39         | 30.00±8.39                  | 20.00                     | 0.272   |
| Headache               | 4 (50.0%)          | 4 (40.0%)                   | 1 (100.0%)                | 0.494   |

Data are presented as mean ± standard deviation or number (percent).

Thirty percent of the women in the study were primiparous. In those with antepartum eclampsia, 30.0% were primiparous and 70.0% were multiparous. The women who had postpartum eclampsia was primiparous. The mean gestational age in women with pre-eclampsia was 36.67±2.52 weeks and in patients with antepartum eclampsia was 34±4.57 weeks.

Figure 1: Clinical presentation with neurological signs and symptoms in antepartum and postpartum patients.

Figure 1 shows the clinical presentation with neurological signs and symptoms in antepartum and postpartum patients. Headache was the most common sign in antepartum and postpartum cases (46.2% and 57.1% respectively, p=0.751). Two antepartum cases had coma (15.4%). 3 cases were disoriented, 3 cases had mono/hemiparesis, and 25.0% had seizure and convulsion (4 antepartum and 1 postpartum).

Figure 2: The number of patients (in percentage) as a function of the computed tomography findings.

The mean systolic blood pressure in the antepartum group was 160±14.72 mmHg and group were 158.57±10.69 mmHg and diastolic being 99.29±15.39 mmHg.

Noteworthy that five patients having eclampsia presented with generalized seizures in our study. None of the patients had recurrent convulsions after starting magnesium sulphate regimen in the hospital.

CT scan of patients showed hypodensities in different regions such as in grey white areas suggestive of brain edema except in 2 patients who had normal scan (Figure...
2). According to Figure 2, the majority of patients had generalized diffuse lesions brain edema while the remaining showed localized lesions bilateral symmetrical parietal-occipital hypodense lesions involving more of white matter than the grey. Some of the patients’ lesions extended to the parietal-temporal areas of both sides but never extended anteriorly beyond the sylvian fissure. Two patients’ lesions could be detected in the basal ganglia and external capsule. These patients had areas of hyper density within the hypodense areas suggesting hemorrhage.

![MRI Findings](image)

Figure 3: The number of patients (in percentage) as a function of the magnetic resonance imaging findings.

MRI of these patients showed bilateral parietal-occipital subcortical white matter hyper density, white matter edema in temporal and parietal-occipital region suggestive of posterior reversible encephalopathy syndrome (PRES) (Figure 3).

Patients with visual disturbances had significant findings on MRI in the parietal-occipital lobes. Bilateral white matter edema in occipital and parietal lobes is suggestive of PRES, hyper dense lesion in occipital and parietal lobes with intratentorial and periventricular hypo-intensification, generalized vasospasm, and increased T2 system in posterior cortical and sub cortical region. These may be due to hypertension induced insult in occipital lobes. MRI features were suggestive of generalized vasospasm, extensive sub cortical white matter hyper intensification in right caudate nucleus, Globus pallidus, putamen, internal and external capsule and supratentorial area in patients with neurological deficit. Neurological evaluation of brain nerves, motor and sensory system was in normal limits. None had signs of meningeal irritation.

**DISCUSSION**

In both pregnancy and puerperium, a number of pathologic manifestations involves the central nervous system. The most common presenting complaint of patients in both pregnancy and peripartum phase is headache. Hypertensive disorders in pregnancy accounts for 14% of maternal mortality worldwide. Peripartum seizures cause significant problems such as maternal and fetal morbidity and mortality.

Ecliptic seizures are unique to pregnancy. Eclampsia has been increasing worldwide with an incidence of 0.28%. Usually, eclampsia may result in PRES. Some neurologic conditions are related to the physiologic modifications in pregnancy such as: eclampsia and reversible cerebral vasoconstriction syndrome.

Eclampsia is defined as seizure or coma associated with pregnancy-induced hypertension. More than 30% of cases are diagnosed postpartum. Most antenatal women who present with tonic-clonic seizures or coma have developed pregnancy-induced hypertension. The majority of patients are diagnosed in antepartum, and more than 70% of the diagnoses are made after 20 weeks of gestation. Around 50-75% of patients present with frontal or occipital headaches that usually precede the attack of seizures, and 20-30% have visual blurring or cortical blindness similar to some patients in the present study.

In a prospective study that was conducted to correlate the neurological presentation with neuroimaging in eclampsia patients, a total of 100 women with antepartum or postpartum eclampsia were included. More than half of the study population was aged between 22 to 25 years and 40% of the women were aged ≤21 years. A similar study from New Delhi reported that majority of patients belonged to age group between 20 to 25 years (60%). The mean age in their study was 22.61±2.72 years. Another study reported the mean maternal age as 23.89 years (range 18-30 years). In the present study, most of the women (60%) presented with age between 19 to 30 years (12 cases) while 8 cases were between 31 and 40 years. The mean age of patients in this study was 28.65±7.16 years. Moreover, 50% of women had postpartum eclampsia while 5% had antepartum eclampsia. These results were also similar to the epidemiological and interventional studies conducted in developing countries such as Zimbabwe and Nepal. The majority of eclampsia cases presented in the third trimester of pregnancy. Around 80% of ecliptic seizures occurred intrapartum or within the first 48 hours following delivery. Rare cases were reported before 20 weeks of gestation or as late as 23 days postpartum.

Moreover, in our study, 30% of the women were primiparous while in the New Delhi study 59% were primiparous. Among them, 64.71% had antepartum eclampsia while 56.06% had postpartum eclampsia. Among multiparous women, 35.29% and 43.94%, had antepartum and postpartum eclampsia respectively.

Both CT and MRI can depict the characteristic imaging features of the neurological disorders in addition to development of complications; hence, leading to early and prompt diagnosis of these disorders and better management of the patients. It is important that the
radiologist be familiar with these entities to be able to evaluate patients efficiently. MRI plays a crucial role in depicting these disorders. MRI is known for its superior soft tissue contrast and multiplane resolution compared to CT. This technique is effective for the diagnosis of hemorrhage and ischemia or edema in preeclampsia. Symptoms such as visual blurring, loss of vision and ophthalmological signs in eclampsia are suggestive of occipital lobe involvement which was supported by CT and MRI which showed that the posterior region of the brain was often affected. MRI abnormalities in eclampsia correlated well with clinical findings in comparison to CT. MRI has an added advantage that it could be done on pregnant patients eliminating the radiation hazards of CT. Moreover, CT failed to show some of the abnormalities that were detected by MRI, hence it is a better option of imaging in eclamptic patients.16 Furthermore, the radiologist plays an important role in proposing the correct diagnosis based on the imaging findings. Therefore, radiologists working in the emergency department must be familiar with these disorders to facilitate the diagnosis process.

CONCLUSION

Several central nervous system disorders can occur during pregnancy and postpartum period. Prenatal care and use of proper diagnostic modalities during pregnancy can prevent many of these neurologic complications. Accurate diagnosis is crucial in acute neurologic disorders during pregnancy and postpartum period since appropriate and timely treatment can reverse the disease process and can reduce the risk of short- and long-term complications.

Failure to diagnose these disorders at the earliest stages can cause life-threatening complications, like ischemia, massive infarction, and death. Common complications that occur are ecliptic encephalopathy, followed by CVT, PRES, and ischemic stroke. The use of appropriate imaging modality aids in the early diagnosis of neurological illnesses consequently help obstetricians to institute appropriate treatment strategies. Future studies with larger sample of pregnant women during and after delivery are needed to further examine the influence of neurological disorders on maternal and fetal outcomes.

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