Case Report

Seizure Disorders in Pregnancy

Srinija.P*., Anoop Sreevalsan**., Abubacker.S. ***.,Swarnapriya .K****., Somasundaram *** , Vasantha N.Subbiah ***** Subbiah.V.K******

* Intern, **Associate Professor, Dept. of Obstetrics and Gynaecology, ***Associate Professor, Radiology, **** Assistant Professor,***** Professor & HOD,Dept. of Obstetrics and Gynaecology, ******Retd.Prof. of Medicine, Chettinad Hospital & Research Institute, Chennai, India.

Dr. Srinija is doing her internship at Chettinad Hospital and Research Institute. Her fields of interest include General Medicine / Obstetrics & Gynaecology.

Corresponding author - Dr. P Srinija (srinijap@gmail.com)

Abstract

Generally seizures occurring in pregnancy are thought to be due to eclampsia. We analyzed patients who were pregnant or in puerperium and presented to our hospital with convulsions, from the years 2007 to 2013. In this period, there were 4315 deliveries in our hospital. We found that 18 patients had convulsions complicating pregnancy & puerperium, an incidence of 0.417%. Of these, 12 were patients had with eclampsia [incidence of 0.27%] and 6 patients had non-eclamptic seizures, with an incidence of 0.139 %. Of these 6 patients, 2 had posterior reversible encephalopathy syndrome. The other 4 cases of non-eclamptic seizures in pregnancy were due to cortical venous thrombosis, subarachnoid hemorrhage & neurocysticercosis. None of these patients had a history of epilepsy. We are presenting these cases to highlight that several other conditions apart from eclampsia, need to be considered in a person without epilepsy presenting with convulsions during pregnancy.

Key Words: Seizure disorders in pregnancy, Cortical venous thrombosis, Subarachnoid hemorrhage, neurocysticercosis

Chettinad Health City Medical Journal 2014; 3(1): 22 - 25

Introduction

Eclampsia is the occurrence of convulsions or coma with hypertension, proteinuria and/or pedal edema between 20 weeks of gestation and 48hrs postpartum. Atypical cases are those that develop at less than 20 weeks of gestation and after 48 hours postpartum and that have some of the signs and symptoms of preeclampsia-eclampsia without the usual hypertension or proteinuria[1]. There are many causes for seizures in pregnancy other than eclampsia, like cerebral infarction, hemorrhage, due to malformations, hypertensive encephalopathy, cerebral venous thrombosis, cerebral malaria, meningitis, intra cranial tumors, metabolic diseases, posterior reversible encephalopathy syndrome, thrombophilia, thrombotic thrombocytopenic purpura[2] etc.,We present 4 such cases of non-eclamptic seizures in pregnancy due to cortical venous thrombosis, subarachnoid hemorrhage & neurocysticercosis.

Case1: Neurocysticercosis

A 29 year old gravida2 para1 live1 presented with history of convulsions at 9 weeks of amenorrhea. She had 1 convulsion at home and 1 at the local hospital. She was given phenytoin sodium at a local hospital and then referred to our hospital. She had caesarean for her previous pregnancy 1 year three months back due to pre-eclampsia. When the patient presented to us, she was drowsy, not oriented to time and place, responding to painful stimuli. Blood pressure on admission was 140/80 mm Hg and pulse rate was 102 bpm. She was afebrile. Cardiovascular system and respiratory system were found to be normal.

Investigations revealed a urine pregnancy test which was weakly positive. Except for an elevation in total count to 16700cells/cmm, uric acid level of 10.8 mg/dl and 1+ albumin in urine, rest of the blood investigations was within normal limits. Ultrasound pelvis showed a single live intra-uterine gestation corresponding to 8 weeks 6 days;MRI showed right frontal granuloma with edema (Neurocysticercosis){Fig.1-4}.

In view of the diagnosis, patient requested a medical termination of pregnancy (patient was counseled on alternative modes of management), which was performed with Mifepristone 200 mg followed by Misoprostol 600 mcg vaginally. She was started on Tablet Albendazole & IV dexamethasone for 3 days followed by Tablet Prednisolone 8 mg for 2weeks. She was discharged and on follow up no further convulsions were reported.

Complicating factors in this case: [1] Previous history of preeclampsia - there is 25% chance of pre eclampsia to recur in subsequent pregnancies (Dutta)[3]. [2] Urine albumin + 1 [3] Raised total count of 16700 [4] Serum uric acid 10.8mg/dl [5] Limitation on imaging procedures due to pregnancy.

Discussion

In the 2 other reported cases (Singhal et al.,) also the urine albumin was raised to 1+4. MRI has again proven itself to be an invaluable diagnostic tool in other reported cases[4,5].

Case2: Sub Arachnoid hemorrhage

25 year old Primigravida, post IUI conception with twin gestation at 31 weeks +3 days presented with preterm labour pains. Patient was conscious and oriented. Blood pressure on admission was 110/60 mm Hg and pulse rate was 76 beats/ min. Cardiovascular system and respiratory system were found to be normal, per abdomen- over distended uterus with multiple fetal parts felt, both fetal heart rates were good, per vaginal examination- no draining / bleeding per vaginum.

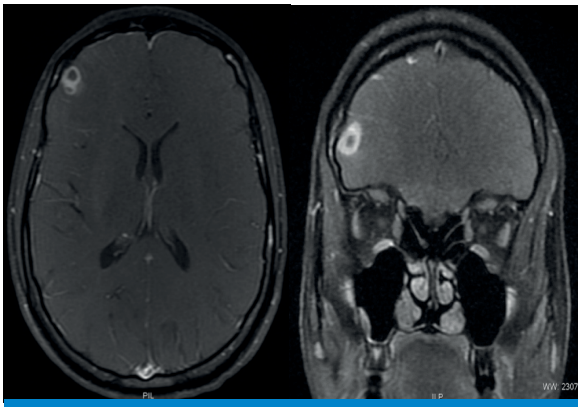

Fig 1 & 2: Contrast T1 Coronal & Contrast T1 Axial : Showing ring enhancing conglomerate lesions in the right frontal lobe.

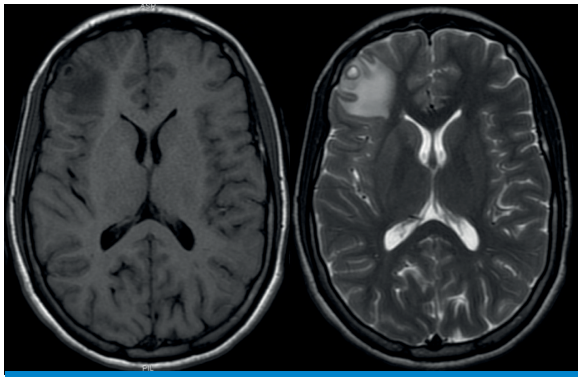

Fig 3 & 4 - T1Weighted &T2 Weighted images: Showing focal lesion with surrounding edema in right frontal lobe in grey-white matter junction.

She was admitted and as steroid prophylaxis was already given before admission, tocolytics were started. The next day she went in for preterm premature rupture of membranes. In view of extreme preterm premature rupture of membranes, patient was taken up for emergency Lower segment caesarean section and Twin A was a girl baby weighing 1.440kg with an Apgar score of 5/10, 8/10, O positive blood group. Twin B was also a girl baby weighing 1.505 kg with an Apgar score of8/10 ,8/10, B negative blood group. There was atonic postpartum hemorrhage which was managed medically and 1 unit of blood was transfused. Anti D was given. Patient developed fever of 101 F after 12 hours postoperatively & was treated with paracetamol. After 16 hours post delivery, patient had 1 episode of tonic clonic convulsions with a blood pressure of 150/84 mm Hg and Pritchards regime (magnesium sulphate regime) was started. Patient was also started on antibiotics. Fundus examination was found to be normal. Blood investigations were within normal limits. Neurophysician advised for a MRI brain / MRV to rule out cortical vein thrombosis.MRI showed intracerebral hemorrhage in left basal ganglia, sub arachnoid hemorrhage in left parietal and bilateral occipital regions. Patient was initially started on IV. Fosphenytoin & Nimodipine. Subsequently, phenytoin and sodium valproate were given orally. CT brain showed resolving subarachnoid hemorrhage. She developed diabetes insipidus which resolved spontaneously. She was discharged on post operative day 29 and advised to follow up with neurophysician.

Complicating factors: [1] history of postoperative fever

Case 3: Cortical venous thrombosis

A 23 year old gravida2 para1 live1 with previous lower segment caesarean section for oligohydramnios and cephalopelvic disproportion, was admitted at 38 weeks 5 days for safe confinement.

Clinical and biochemical examination of the patient were normal. Patient developed labor pains and was taken up for an emergency repeat lower segment caesarean section. She delivered a girl baby weighing 3.355 kg with an Apgar score of 9/10. On the 5thpost operative day, patient suddenly had one episode of generalized tonic clonic convulsions with a BP of 150/100 mm Hg, and was treated with IV Phenytoin. She had 3 more episodes of generalized tonic clonic convulsions and was given loading dose of IV Phenytoin and was shifted to ICU and intubated. CT brain revealed Right frontal hypodensity with small area of hyperintensity. Patient was diagnosed to have cortical vein thrombosis and was put on Intravenous heparin, dexamethasone, mannitol, torsemide, levetiracetam, sodium Valproate, and midazolam. MRI showed features suggestive of cortical venous thrombosis with acute hemorrhagic infarct in left frontal lobe. Patient subsequently improved and was discharged.

Case 4: Cortical venous thrombosis

26 year old female was brought in by her relatives with history of unresponsiveness for duration of one hour. Patient had delivered a full term girl baby through normal vaginal delivery at a local primary health centre 15 days ago, following which she had persistent vomiting & abdominal pain. She also had intermittent fever for the past 15 days. Patient developed numbness of right upper and lower limbs after admission along with 1 episode of generalized tonic clonic convulsions.

Patient was unconscious, pulse rate was 82bpm, blood pressure on admission was 110/60 mm Hg. Cardiovascular and respiratory systems were found to be normal. Per abdomen was soft. Both pupils were equal and reacting to light. There was right facial lag, catatonia of left upper limb, paucity of movements in right upper & lower limbs on painful stimuli and bilateral plantar flexor. All deep tendon reflexes were present in upper limb and brisk in lower limbs.

Blood work up and USG Doppler to rule out deep vein thrombosis did not reveal anything abnormal. CT brain showed cerebral vein thrombosis with hypointensities in left temporal and right frontal regions probably due to Thrombosis. MRI brain showed features suggestive of cortical venous sinuses thrombosis.

Patient was admitted in the ICU and was started on Intravenous Heparin, Dexamethasone, Mannitol, Ceftriaxone, Levetiracetam and Phenytoin. Then Inj.Heparin was changed to Inj.Enoxaparin. Patient progressively deteriorated and expired 5 days after admission. The cause of death was cortical venous sinuses thrombosis with malignant intracranial hypertension with severe brainstem dysfunction.

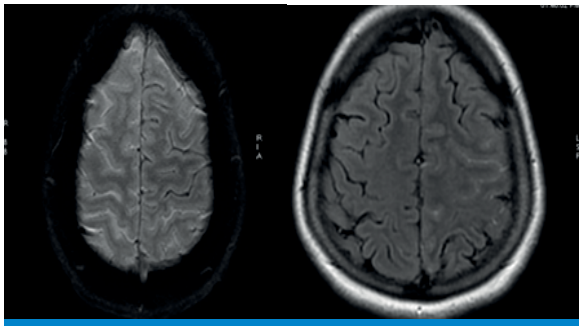

Fig 5 & 6 - T2 FLAIR & GRE images:
Showing linear hyper intensities noted on T2 FLAIR image with GRE blooming in the left parietal region, suggestive of subarachnoid hemorrhage.

Complicating factors: [1] history of postoperative fever

Discussion

This article highlights the fact that all convulsions in pregnancy are not due to eclampsia. We found that we had 18 cases of convulsions complicating pregnancy & puerperium giving us an incidence of 0.417%. Of these 12 were cases of eclampsia [an incidence of 0.28 %] and 6 were cases of non-eclamptic seizures [an incidence of 0.14 %]. Of these 6 patients, 2 had posterior reversible encephalopathy syndrome[6,7].

Table - 1

	No. of cases	Incidence
All convulsions	18	0.417
Eclampsia	12	0.28
Non eclamptic convulsions	6	0.14

Fig 7 & 8 T2 FLAIR & GRE images:
Showing mixed intense area noted in left frontal lobe in T2flair, blooming noted in left frontal lobe in GRE.

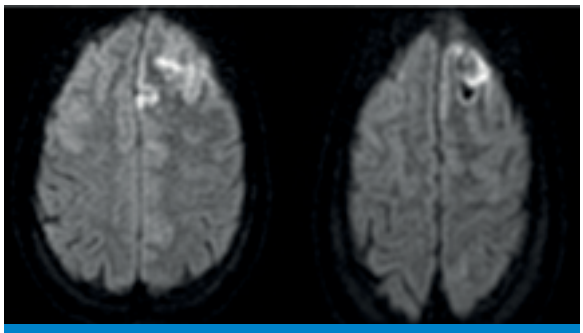

Fig 9 & 10 - DWI images:
Showing diffusion restriction in the left frontal lobe

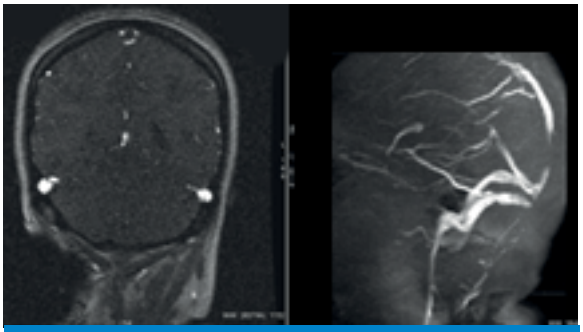

Fig 11 & 12 MRV :
Showing thrombus/ no flow in superior sagittal sinus

The differential diagnosis that should be considered include:

[1] Epilepsy: incidence is around 1 in 200 pregnancies. Usually previous history of epilepsy can be elicited.

[2] Infections – encephalitis & meningitis – fever is usually present, increased total counts are seen. Neurocysticercosis & tuberculoma are other infections that usually present as a space occupying lesion. Neurocysticercosis is due to the cysts of Taenia solium the pork tapeworm[8]. Infection happens when humans consume the ova from exogenous sources or through self infection by the fecal-oral route.

These then are digested in the stomach and release oncospheres which penetrate the intestinal wall and reach the blood stream. These oncospheres develop into cysticerci in any organ. In countries where it is endemic, cysticercosis may affect 2-4% of the populations. Convulsions in pregnancy have different consequences than in the non-pregnant. The hypoxia and acidosis caused by the convulsions, although well tolerated by the mother, can be fatal to the fetus[5].

The treatment of choice for neurocysticercosis during pregnancy is Albendazole and Prednisolone. If the patient has an intraventricular cyst, then post delivery shunting is advisable[3].

[3] Stroke – either hemorrhagic or ischemic. Ischemic strokes are caused due to cerebral vessel thrombosis or cerebral embolism. These include cortical venous thrombosis.

Hemorrhagic strokes are due to either intracerebral hemorrhage or subarachnoid hemorrhage. In pregnancy the intracerebral haemorrhage is usually associated with hypertension superimposed on pre-eclampsia or occasionally pure pre-eclampsia. Intracerebral haemorrhage has a higher mortality rate than subarachnoid haemorrhage because of its location. Subarachnoid haemorrhage is bleeding into subarachnoid space, the area between the subarachnoid membranes and pia mater surrounding the brain.

Causes: Berry aneurysms, arterio- venous malformations, infections, coagulation disorders, angiopathies, venous thrombosis, drug abuse (cocaine), tumours and trauma.

Subarachnoid haemorrhage occurs at a frequency of 6 in 1, 00, 000 pregnancies. The incidence does not differ from the general non-obstetrical population but the mortality during pregnancy can be as high as 35%. Idiopathic subarachnoid haemorrhage constitutes about 15-30% of sub arachnoid hemorrhage.

The classical signs and symptoms are:

1. Thunderclap headache,
2. Fever (around 102.2 degree F),
3. Vomiting,
4. Altered consciousness,
5. Papilloedema, 6. Neck stiffness,
7. Seizures (1 in 14),
8. Terson's syndrome (subhyaloid haemorrhage).

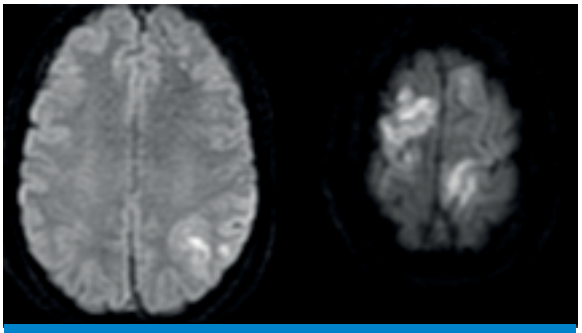

Fig 13 & 14 DWI images:
Showing multiple acute infarcts in fronto-parietal region

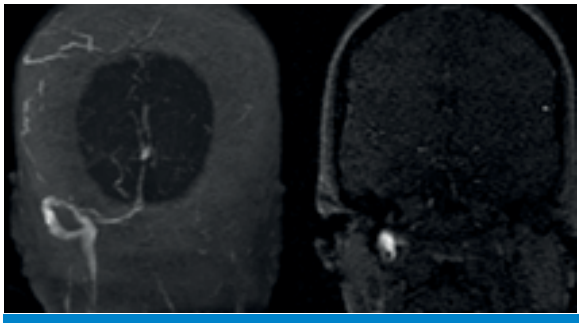

Fig 15 & 16 MRV:
left transverse sinus and superior sagittal sinus are not visualized. Right transverse sinus and sigmoid sinus are thinned out.

[4] Posterior reversible encephalopathy syndrome (PRES) - clinical features include headache, nausea, vomiting, seizures, visual disturbances and altered sensorium. PRES is due to failure of cerebral auto regulation and endothelial dysfunction. It is essentially a clinico-radiological diagnosis[2]. In PRES, the MRI images show hyper intense lesions in the cerebral white matter mainly in the parieto-occipital region. Occasionally the lesions involve the grey matter[6].

Cortical venous thrombosis is a rare disorder affecting 5/1000000. The prevalence of cortical venous thrombosis in the Indian population is about 4.5 / 1000 obstetric admissions[6]. Our incidence is 0.46/1000. Similarly the case fatality rate is less than 10%[9].

Acknowledgements

We wish to thank our Dean Prof.Ravindran for his support and encouragement. We express our gratitude towards Dr.Subramanian, Consultant Neurophysician as well as the department of radiology for their valuable inputs. We also are grateful to the Medical Records Department for their patient cooperation and for providing us with the material required for this paper.

References

1) Sibai BM, Stella CL, Diagnosis and management of atypical preeclampsia-eclampsia, AmJ Obstet Gynecol 2009; 200:481.e1-481.e7

2) Mustafa Albayrak, İsmail Özdemir, Yavuz Demiraran, Süber Dikici, Atypical preeclampsia and eclampsia: report of four cases and review of the literature in J Turkish-German Gynecol Assoc 2010; 11: 115-7

3) D.C.Dutta, Hypertensive disorders in pregnancy, Textbook of Obstetrics, 7th edition 2014: 219-40

4) Singhal et al.: Neurocysticercosis as an important differential of seizures in pregnancy: two case reports. Journal of Medical Case Reports 2011; 5:206.

5) Gowri.V,Jacob.P.C,Jain.R,Sawhney.K,"Neurocystic ercosis in pregnancy" Neurosciences 2005; Vol.10 (2): 183-185

6) Srinija.P., Abubacker Sulaiman., Anoop Sreevalsan, Somasundaram, Vasantha N.Subbiah. "Posterior reversible encephalopathy syndrome "Chettinad Health City Medical Journal 2013; 2(2): 65-68.

7) Kadkol.R, Godbole.R.R, ante partum eclampsia with posterior reversible encephalopathy syndrome, Journal of Obstetrics and Gynecology Societies of India 2012; 62 (s1): s27-s28

8) Suarez, Victor R.,Thomas A. Iannucci. Neurocysticercosis in pregnancy: a case initially diagnosed as eclampsia. Obstetrics & Gynecology 93.5, Part 2 1999: 816-818.

9) Kakade.A.S & Kulkarni.Y.S "Cerebral venous thrombosis in pregnancy" Bombay Hospital Journal, 2011; Vol53 Special issue