MATERNAL AND FETAL OUTCOME IN EPILEPSY COMPLICATING PREGNANCY

DISSERTATION SUBMITTED TO

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M.D. OBSTETRICS AND GYNAECOLOGY

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MADURAI

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DECLARATION

I ***Dr. P. JEYARANI*** *solemnly declare that the dissertation titled '****Maternal and fetal outcome in epilepsy complicating pregnancy'*** *has been prepared by me. I also declare that this Bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other university board either in India or abroad.*

This dissertation is submitted to the ***Tamil Nadu Dr.M.G.R Medical University****, Chennai in partial fulfillment of the rules and regulation for the award of* ***M.D Degree, Branch –II (Obstetrics and Gynaecology)*** *to be held in October 2011.*

Place*: Madurai*

Date*:* ***(Dr. P. JEYARANI)***

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled ***'MATERNAL AND FETAL OUTCOME IN EPILEPSY COMPLICATING PREGNANCY'*** *is a Bonafide work done by* ***Dr. P.JEYARANI,*** *under my direct supervision and guidance, submitted to the* ***TAMIL NADU Dr.M.G.R MEDICAL UNIVERSITY,*** *in partial fulfillment of university regulation for M.D degree, Branch- II, (Obstetrics and Gynaecology).*

Signature of Head of Department

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I wish to thank all patients who participated in this study.

CONTENTS

INTRODUCTION

Epilepsy describes a condition in which a person has recurrent seizures due to chronic underlying process. Epilepsy refers to a clinical phenomenon rather than a single disease entity, since there are many forms and causes of epilepsy.

Traditionally, the diagnosis of epilepsy requires the occurrence of at least two unprovoked seizures which are 24 hours apart. Some clinicians diagnose epilepsy when one unprovoked seizure occurs in the setting of an interictal discharge. Seizures are the manifestation of abnormal hypersynchronous discharges of cortical neurons. The signs and symptoms of seizures depend on the cortical location of the epileptic discharges and the propagation pattern of the epileptic discharge in the brain.

The causes of epilepsy may primary or secondary. Epilepsy is usually managed by neurologists or general practitioners. Treatment is symptomatic and similar whether the epilepsy is primary or secondary with usage of antiepileptic drug.

Epilepsy is not a contraindication to pregnancy. Women with epilepsy can be reassured that having epilepsy should not prevent them from having children. However close medical care is essential and a multidisciplinary approach is recommended.

Pregnancies in women with epilepsy are high risk and need careful management by both the medical and obstetric teams due to the increased incidence of complications and adverse outcomes of pregnancy. By the time a pregnant woman with epilepsy

presents, the fetus is virtually fully formed and the opportunity for altering drug treatment has passed (Crawford P., 2002)[6]

Majority of women with epilepsy will have normal and healthy infants. Effective pre-conceptional counseling and medical care is essential for the treatment of the pregnant women with epilepsy. Exposure to AED has been associated with two to three times increase in major malformations in infants exposed in utero as compared to the ordinary population. Managing epilepsy during pregnancy is a major therapeutic challenge, as the potential adverse effects of antiepileptic drugs on the fetus must be balanced against the maternal and fetal risks associated with uncontrolled seizures (Torbjörn Tomson, Vilho Hiilesmaa, 2007)[18]

It is very important that all women with pregnancies have a preconception evaluation done by a neurologist, when the need to continue AEDs or possibility of reducing AED load could be assessed (Thomas SV, 2011). [17].

EPILEPSY

Epilepsy - (from the Ancient Greek ***epilēpsía***) recognized from the dawn of history as 'disease of lightening' and it was correctly described by JH. Jackson little over a century ago.

Epilepsy is a neurological disorder characterized by paroxysmal cerebral dysrhythmia, manifesting as brief episodes (seizures) of loss / impairment of consciousness, abnormal motor, sensory or psychiatric phenomena, with or without characteristic body movements (convulsions).

Epilepsies have been classified as **Generalized** and **Partial** seizures. Generalized seizures include:

1) Generalized tonic-clonic seizures (GTCS / Grand mal, commonest)

2) Absence seizure (petit mal, common in children)

3) Atonic seizures (Akinetic epilepsy)

4) Myoclonic seizures (shock like contraction of muscles)

5) Infantile spasms (probably not a form of epilepsy)

Partial seizures include:

1) Simple partial seizures (SPS / Cortical focal epilepsy)

2) Complex partial seizures (CPS / psychomotor / temporal lobe epilepsy)
   - with or without secondary generalization.

Generalized seizures:

These involve both brain hemispheres simultaneously and may be preceded by an aura before an abrupt loss of consciousness. There is strong hereditary component. In grandmal seizures, loss of consciousness is followed by tonic contraction of the muscles, rigid posturing and gradual relaxation. Return of consciousness is gradual and the patient may remain confused and disoriented for several hours. Absence seizures also called petit mal seizures, are form of generalized seizures that involve brief loss of consciousness without muscle activity and are characterized by immediate recovery of consciousness and orientation.

Partial seizures:

These originate in one localized area of the brain and affect a corresponding neurological function. They are believed to result from trauma, abscess, tumors or perinatal factors, although specific lesion is rarely demonstrated. Simple motor seizures start in one region of the body and progress toward other ipsilateral areas of the body producing tonic and then clonic movements.

Simple seizures can affect sensory function or produce autonomic dysfunction or psychological changes. Consciousness is usually not lost and recovery is rapid. Partial seizures can secondarily generalize producing loss of consciousness and convulsions. Complex partial seizures also called temporal lobe or psychomotor seizures usually involve clouding of consciousness.

Investigations

Epilepsy is one of the commonest neurological disorders in our country. Patients with epilepsy are investigated with EEG, CT and MRI scan.

The **Electroencephalograph (**EEG) which records the electrical activity of the brain cells is helpful, when it is clearly abnormal. But 40-50% of patients with epilepsy have a normal single inter-ictal EEG. On the other hand, about 5% of non-epileptic patients may have non-specific EEG abnormalities. Despite its limitations, the EEG is a simple non-invasive and relatively inexpensive test that gives useful information if used judiciously and correlated with the clinical description of seizures. When abnormal, it is helpful in making a correct diagnosis of epilepsy and may even help in the choice of anti-epileptic drug therapy.

Computed tomography (CT) scanning is helpful, especially where an underlying pathology is suspected as the cause of seizures. Magnetic resonance imaging (MRI) is now established to be a better and safer diagnostic modality than CT scanning for the

detection of an epileptogenic focus or suspected brain abnormalities in patients with seizures.

Causes of epilepsy

Most cases are **primary** (idiopathic) and some may be **secondary** to intracranial tumors, infection (tuberculoma, neurocysticercosis), ischemia, trauma. The diagnosis of idiopathic epilepsy is one of exclusion.

In practice, it is useful to consider the etiologies of seizures based on the age of the patient. During the **neonatal period and early infancy**, potential cause includes hypoxic-ischemic encephalopathy, congenital CNS malformations, CNS infection and metabolic disorders. The most common seizures arising in **late infancy and early childhood** are febrile seizures, which are seizures, associated with fevers but without evidence of CNS infection or other defined causes. **Childhood** mark the age at which many of the well-defined epilepsy syndromes present. Temporal lobe epilepsy usually present in childhood and may be related to mesial temporal lobe sclerosis.

The period of **adolescence and early adulthood** is one of transition during which the idiopathic or genetically based epilepsy syndromes become less common, while epilepsies secondary to acquired CNS lesions begin to predominate. It may be associated with head trauma, CNS infections, brain tumors, and drug / alcohol withdrawal. The causes of seizures in **older adults** include cerebrovascular disease, cerebral tumors and degenerative diseases.

Treatment of epilepsy

Treatment is symptomatic and similar whether the epilepsy is primary or secondary. Varieties of antiepileptic drugs (AED) are in use like phenobarbitone, phenytoin, carbamazepine, valproic acid, ethosuximide, diazepam/clonazepam, lamotrigine and newer drugs like vigabatrin, topiramate & levetiracetam, with each drug having its own advantage and limitations.

EPILEPSY IN PREGNANCY

Pregnancy of women with known epilepsy refer to 'Epilepsy in pregnancy'. The most common cause for seizures during pregnancy is pre-existing epilepsy, while new-onset seizures in latter half of pregnancy or during the immediate postpartum period are most frequently caused by eclampsia.

Some women with epilepsy may experience seizure only during pregnancy which is termed 'gestational epilepsy' such women would be seizure free between pregnancies. Another subgroup (Gestational onset Epilepsy) may have their first seizure during pregnancy and thereafter may continue to get spontaneous recurrent seizures (Charu Jandial., et al, 2007) [4].

Epilepsy is the second common chronic neurological disorder complicating pregnancy after migraine (Chattopadhyay Nibedita., et al, 2008) [5]. About 2.7 million women in India suffer from epilepsy, with 52% of them being in the reproductive age group (Thomas SV 2011) [17]. Incidence of seizure disorder in women attending antenatal clinics is estimated to be 0.3-0.5% of all births. [5, 17]

Pregnancy in the woman who has epilepsy raises several concerns about the risk of adverse maternal and fetal outcomes, including the risk of more frequent seizures and the potential for antiepileptic drug-related teratogenicity (Martha j. Morrell -2002) [12].

The cause of this teratogenicity could be due to direct drug toxicity, drug-induced folate deficiency or genetically determined lack of epoxide hydrolase or free radicals. The role of the hepatic mixed function oxidase system may be specially important in conferring teratogenic risk (Charu Jandial., et al, 2007) [4].

The most important goal of therapy, both prenatally and during pregnancy, is to optimally control seizures. The drug selected for a patient should be determined by the type of seizure she has, using only a single drug if possible.

Regarding the general management of epileptic women the first and foremost is to determine whether a woman of childbearing age requires antiepileptic drugs (Charu Jandial., et al, 2007) [4]. If needed as far as possible monotherapy should be the aim. Diet before conception should contain adequate amounts of folate. Free or plasma levels of AEDs should be regularly checked and they need one antenatal ultrasonography to rule out congenital malformations.

Polytherapy involving treatment with more than one AED also seems to be associated with an increased risk of birth defects compared with monotherapy (Battino., etal, 2007) [2].

EFFECTS OF EPILEPSY ON PREGNANCY

Both published and unpublished data indicate women who have epilepsy face a higher risk of pregnancy-related complications including hyperemesis, anemia, antepartum / postpartum hemorrhages, placental abruption, preeclampsia, premature birth and low birth weight baby. Data on first trimester losses, premature rupture of membranes (PROM), operative vaginal delivery and caesarean section are inconclusive.

Regarding fetus, increased incidence of intrauterine growth retardation (IUGR), cognitive dysfunction, microcephaly and perinatal mortality has been reported with increased incidence of congenital malformations (Page B. Pennell, 2002) [14].

Women with epilepsy have a 4 to 8 percent chance of giving birth to a child with a major malformation, compared with 2 to 4 percent in the general population (Chattopadhyay Nibedita., et al, 2008) [5] (Martha j. Morrell -2002) [12]. Although the maternal trait of epilepsy may predispose neonates to birth defects, a recent study suggests that the risk of birth defects is entirely associated with maternal use of antiepileptic drugs.

Teratogenicity of main anticonvulsants (Charu Jandial., et al, 2007) [4]

1. Phenobarbitone: Probably no rise in malformation rate.
2. Phenytoin: Orofacial clefts, congenital heart defects dysmorphic facial features, fetal hydantoin syndrome.
3. Carbamazepine: Craniofacial defects, finger nail, hypoplasia, developmental delay.
4. Valproic acid: Neural tube defects.

Malformations associated with exposure to the older antiepileptic drugs include cleft lip/palate and ventricular septal defect. Neural tube defects are associated with exposure to valproate and carbamazepine (Tegretol) at a frequency of 1 to 2 percent and 0.5 to 1 percent, respectively (Martha j. Morrell, 2002)[12].

Minor congenital anomalies affect 7 to 15 percent of infants exposed to antiepileptic drugs, which represents a twofold increase over that in the general population. These anomalies principally involve the face and digits, including hypertelorism, epicanthal folds, broad nasal bridge, elongated philtrum, distal digital, and nail bed hypoplasia (Chattopadhyay Nibedita., et al, 2008) [5] (Martha j. Morrell, 2002)[12].

There are several potential mechanisms for antiepileptic drug-mediated teratogenesis. The older generation of antiepileptic drugs generates free radical metabolites and induces folic acid deficiency (Martha j. Morrell, 2002)[12]. The risk of teratogenicity is significantly increased in women taking multiple antiepileptic drugs and in those on high doses of antiepileptic medication. The newer generation of antiepileptic drugs is not teratogenic in animals, but there is no sufficient data in human pregnancy.

Epilepsy in pregnancy presents a unique challenge for both the mother and her baby. Managing epilepsy during pregnancy is a major therapeutic challenge, as the potential adverse effects of antiepileptic drugs on the fetus must be balanced against the maternal and fetal risks associated with uncontrolled seizures.

EFFECTS OF PREGNANCY ON EPILEPSY

The effect of pregnancy on epilepsy are unpredictable and the seizure frequency may increase, decrease or may remain unchanged. Seizure remain unchanged in more than half of women, one third experience more frequent seizures and minority improved.

Seizure frequency **may increase** due to, (Lara E. Jeha 2005) [11]

- Enhanced metabolism & increased drug clearance associated with pregnancy, resulting in decreased serum drug concentration.
- Increased volume distribution of the AED.
- Increased serum binding proteins.
- Decreased or non-compliance with medication.
- Factors which lower the threshold for seizures like sleep deprivation, hormonal changes of pregnancy (high E), associated psychological and emotional stress of pregnancy.

Seizure frequency **may decrease** due to

- Improved compliance with drug regimen in some patients.

In some patients the seizure frequency **may remain unchanged**.

AIM OF THE STUDY

- To evaluate the effect of epilepsy on pregnancy regarding the maternal and fetal outcome.

- To evaluate the effect of pregnancy on epilepsy regarding the frequency of seizure occurrence.

REVIEW OF LITERATURE

(Thomas SV et al., 2001)[16] in his study on Pregnancy in women with epilepsy followed up 85 women with epilepsy for reproductive functions under the registry of Kerala. 32 of them had completed the pregnancy. Pregnancy ended as spontaneous abortion in one patient. Nearly one third required cesarean section. Majority (87.5%) had term babies and three (10.7%) babies had birth asphyxia. Six babies (21.4%) had low birth weight. Congenital malformations were detected in four cases (12.5%). The risk of malformation was significantly greater ($p<0.05$) when the mother had generalized epilepsy. Majority of women with epilepsy had safe pregnancy and childbirth without any aggravation of epilepsy.

(Page B. Pennell., 2002)[14] in his article in seminars in neurology about Maternal and Fetal outcomes in pregnant women with epilepsy has summarized epilepsy is associated with increased obstetric risks and increased adverse neonatal outcomes. Prior to conception, folic acid should be administered and the antiepileptic drug (AED) regimen should be optimized, preferably using AED monotherapy. Folate supplementation should begin prior to conception and is crucial during the first 30 days of gestation. Periodic monitoring of total and free AED levels is recommended. Prenatal screening should be offered. Careful planning and management of any pregnancy in women with epilepsy are essential to increase the likelihood of a healthy outcome for the mother and infant.

(Crawford P., 2002)[6] in his study on Epilepsy and pregnancy had indicated that all major anticonvulsant drugs are teratogenic but the main risk to the developing fetus appears to be when the mother is on polytherapy especially if sodium valproate forms part of the combination. Folate supplements (5 mg) before conception are advisable. He also denoted that there appears to be a minor but significant increased risk of maternal complications such as hyperemesis gravidarum, pre-eclampsia and eclampsia, vaginal bleeding and premature labour. In the majority of women seizure control will not alter during pregnancy. There is an increased risk of prematurity (9-11%), stillbirth, neonatal and perinatal death, haemorrhagic disease of the newborn, low Apgar scores and low birth weight (7-10%).

In a retrospective case-control study done by (P. Goel, L. Devi et al., 2006) [9]for Maternal and Perinatal Outcome in Pregnancy with Epilepsy, they had concluded, though significant proportion of the epileptic patients had seizures during pregnancy, the maternal and fetal outcome was similar in both the groups. There was no statistically significant difference in the pregnancy related complications such as pregnancy induced hypertension (PIH), eclampsia, abruptio placenta, placenta previa, anemia and gestational diabetes mellitus (GDM) in both the groups. Mean period of gestation and rate of caesarean section was similar in both the groups. Prematurity was observed in 21.6% cases and 17.6% in controls.

Fetal outcomes (APGAR score, birth weight, still births, neonatal death and congenital malformation) were also similar in the two groups. 75% of the patient in study group were on monotherapy. Fourteen patients had seizure during pregnancy, but there were no maternal complications.

(Battino, Dina; Tomson, Torbjörn., 2007)[18] in their neurology article for Management of Epilepsy during Pregnancy had stated that the teratogenic effects of valproic acid appear to be dose dependent, with higher risks at dosage levels >1000 mg/day. Polytherapy involving treatment with more than one AED also seems to be associated with an increased risk of birth defects compared with monotherapy. They advised to use the appropriate AED as monotherapy in the lowest effective dosage throughout pregnancy in such a way that seizures are avoided but with minimized risks to the fetus and to avoid valproic acid if possible. Any major change in the treatment of a woman with epilepsy should ideally be completed before conception and they recommended regular monitoring of drug concentrations during pregnancy.

A prospective study done by (Chattopadhyay Nibedita et al., 2008)[5] for Feto-maternal outcome in pregnancy with epilepsy, had stated that out of 43 pregnant women they studied, fetal loss and major malformations were observed in 4.65% cases each. Out of the 41 live born infants major congenital malformations were observed in two babies and minor anomalies in three.

A population-based cohort study done by (Borthen I et al., 2010)[3] for delivery outcome of women with epilepsy had concluded that pregnant women with epilepsy have a low complication rate. However they have a slightly increased risk of induction, caesarean section and postpartum haemorrhage. These rates were even higher for women with epilepsy and antiepileptic drug use. In addition, the risk of an Apgar score < 7 was higher. He also suggested it is not possible to ascertain on the basis of this study whether this is a result of more severe epilepsy or antiepileptic drug use.

(Sanjeev V Thomas, 2011)[17] in his article of Managing epilepsy in pregnancy has stated about 10% of the babies in pregnant women with epilepsy may have major congenital malformations and most adverse outcomes are related to antiepileptic drugs (AEDs). Polytherapy, high dose of any AED, traditional AEDs like phenobarbitone /sodium valproate are probably associated with higher risk fetal complications. They had also advised it is important that all women with epilepsy should have a preconception evaluation done by a neurologist regarding the AED and to take folic acid 5 mg daily during preconception period and pregnancy. They should undergo a detailed screening for fetal malformations between 12 and 18 weeks of pregnancy.

MATERIALS AND METHODS

This prospective study on epilepsy complicating pregnancy was conducted in Government Rajaji hospital (GRH), Madurai from June 2010 to May 2011. Permission from Ethical Committee of the hospital was obtained for research purpose.

All consecutive epileptic patients admitted in department of Obstetrics and Gynecology**,** Government Rajaji Hospital, Madurai, were recruited for this study with fulfillment of following criteria.

Inclusion criteria

1. All pregnant women with history of epilepsy
2. Both primigravida and multigravida
3. Both booked and unbooked cases
4. All type of seizure disorder (GTCS, Partial)
5. Patients on regular or irregular intake of AEDs
6. Patients on Monotherapy or polytherapy of AEDs
7. Patients not on AEDs as per neurologist advice
8. Patients who discontinued AEDs by themselves

Exclusion criteria

1. Eclamptic patients
2. Postpartum seizure (including Eclampsia, CVT)
3. Cases of metabolic encephalopathy / drug toxicity
4. Psychogenic causes

Pregnant women with history of epilepsy, recruited for this study were enquired with detailed history regarding their age, booking status, gravida and details of menstrual history to arrive expected date of delivery. Their detail obstetric history was recorded especially about their performance during previous delivery. History of imminent symptoms like headache, nausea, vomiting, blurring of vision, epigastric pain and oliguria was obtained. History of other obstetrical complications like oligohydramnios, PIH, antepartum hemorrhage was also obtained.

Past history regarding the age of onset of seizures, type of seizure, period at which the patient had last seizure, disease free interval and history of status epilepticus was obtained. The details of antiepileptic drugs, type and duration of therapy, regular or irregular intake of AED and folic acid intake was obtained.

Presence of other medical conditions like diabetes mellitus, hypertension, tuberculosis, heart disease and renal disease were elicited. Detail neurological history to rule out CNS lesion, trauma, infection, and tumor and drug intake was elicited.

General and systemic examination including thorough CNS examination, per abdominal and per vaginal examination was done for all patients. Blood pressure measurement and fundus examination was also done. Neurologist opinion was obtained for all cases.

Investigations like HB%, urine (Alb, sugar) blood urea, sugar, serum creatinine, serum uric acid, serum electrolytes, liver function tests and platelet count were done. EEG was taken for all cases.

Ultrasonographic examination was done for gestational age assessment, liquor status, IUGR and to rule out congenital anomalies.

All pregnant epileptic women were admitted in ward before the expected date of delivery and evaluated for epilepsy and their treatment was individualized according to neurologist advice. As the patient gets into labour, they were monitored carefully during labour with partogram and the mode of delivery in all patients was recorded. Caesarean section was done for obstetric indications.

Maternal outcome was evaluated by the occurrence of seizures during labour or postpartum and for other obstetric complications like oligohydramnios, PIH, antepartum haemorrhage, postpartum haemorrhage.

Fetal outcome was obtained by the Birth weight of the baby, Apgar scoring and evidence of any congenital anomalies. Apgar scoring was recorded for 1 and 5 minutes. Pediatric opinion was obtained for all neonates. Neonate with prematurity, low Apgar scores, IUGR, and anomalies were admitted in Neonatal intensive care units. Head circumference measurement was done for all babies.

All delivered epileptic patients were observed in the labour ward for period of 48 hours following delivery. Postnatally all patients were subjected to CT to rule out other causes. AEDs were continued postnatally and patients were discharged after obtaining neurologist advice. Postnatal counseling done to continue the AEDs.

Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart.

Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2010)** developed by Centre for Disease Control, Atlanta.

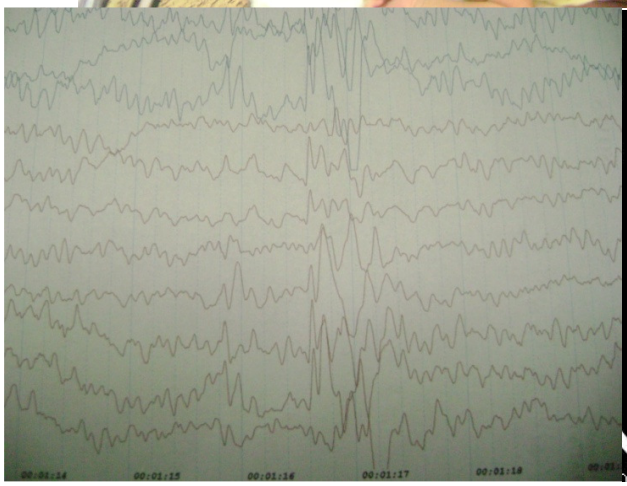
Using this software range, frequencies, percentages, means, standard deviations, chi square and ‘p’ values were calculated.

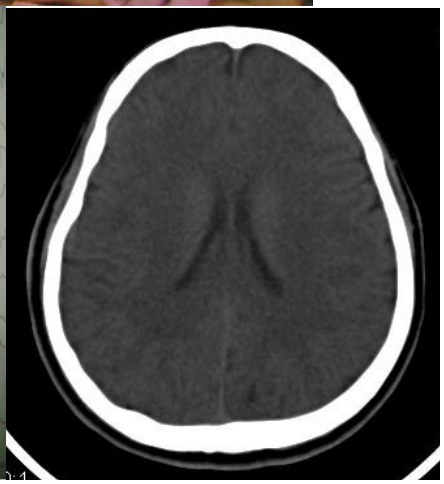
Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables.

A 'p' value less than 0.05 is taken to denote significant relationship.

REPRESENTATIVE CASES

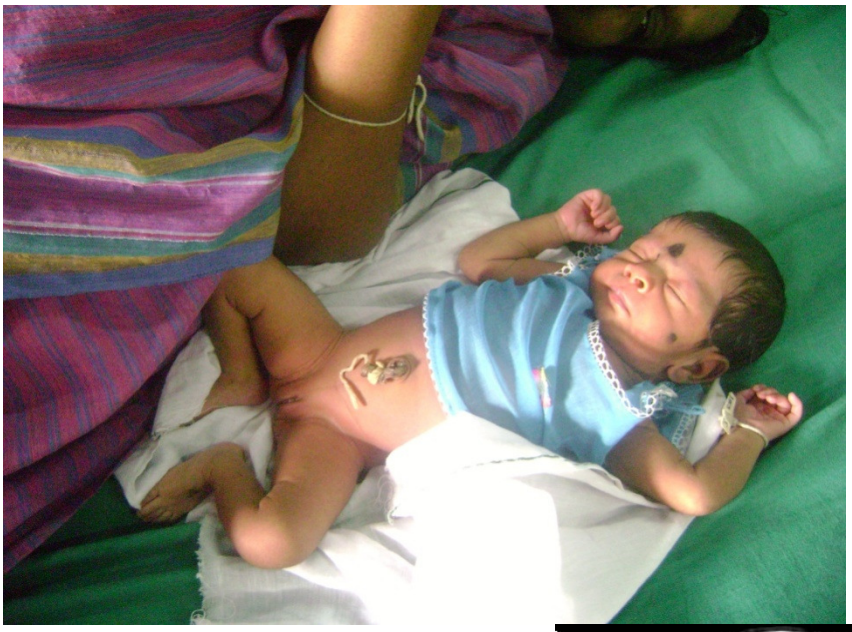
Case: 1

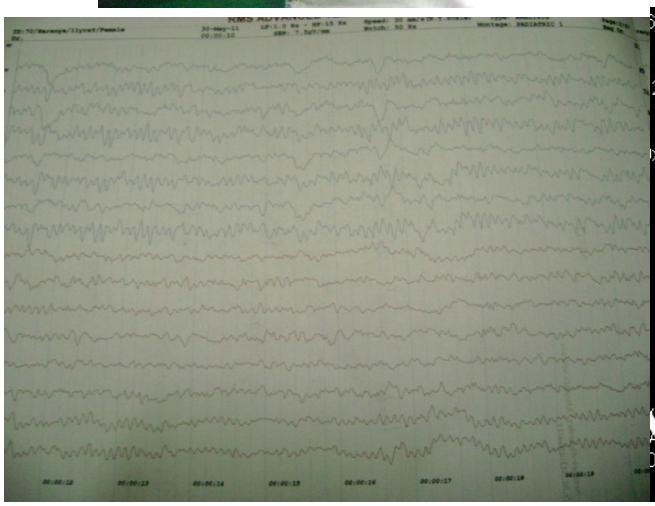


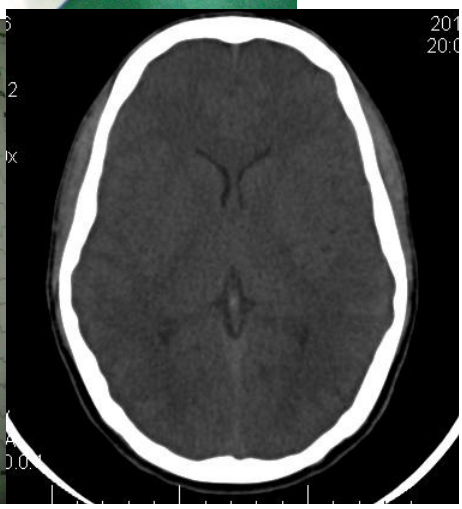


20 year old primigravida with GTCS, last fit 4 years back. She had abnormal EEG and normal CT. She was on regular intake of T. Phenytoin 2Hs and folic acid and she delivered a term live male baby with birth weight of 2.7 kg with good Apgar without any maternal complication

Case 2:



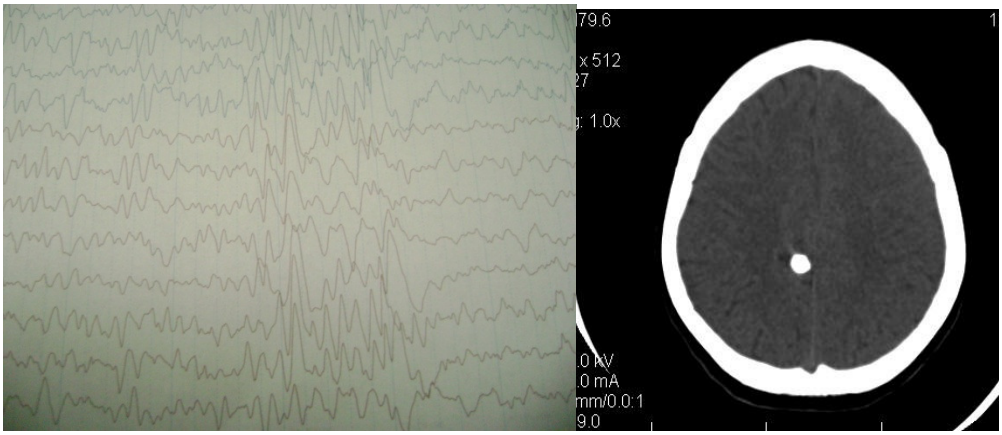




25 year old primigravida with GTCS and disease free interval for 2 months. She had normal EEG and normal CT. She was on regular intake of T. carbamazepine 2-1-2. LSCS done for failed induction with fetal distress. New born Birth weight was 3 kg with good Apgar score.

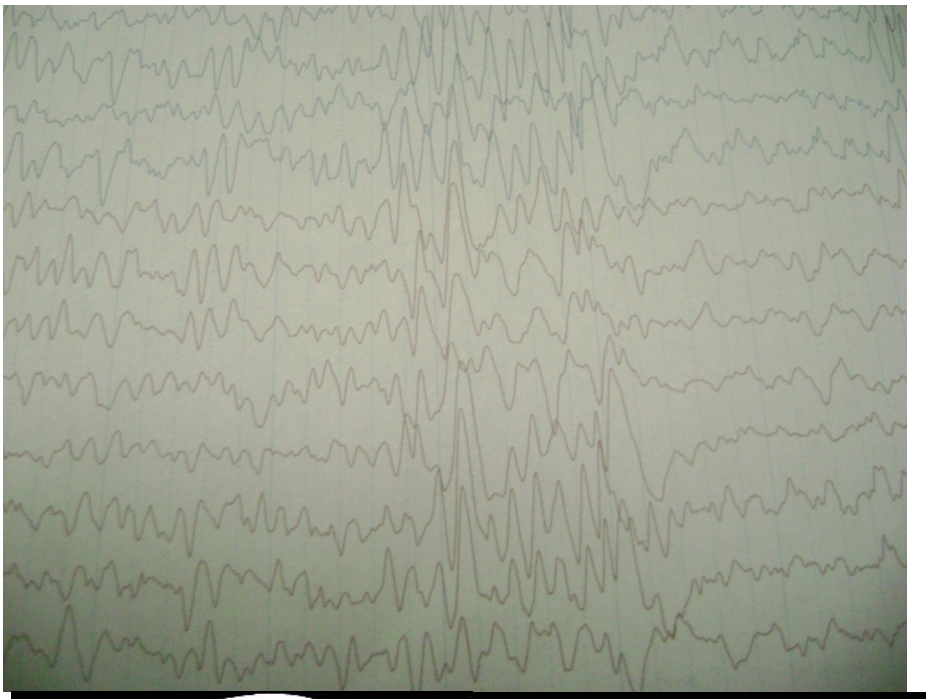
Case 3:

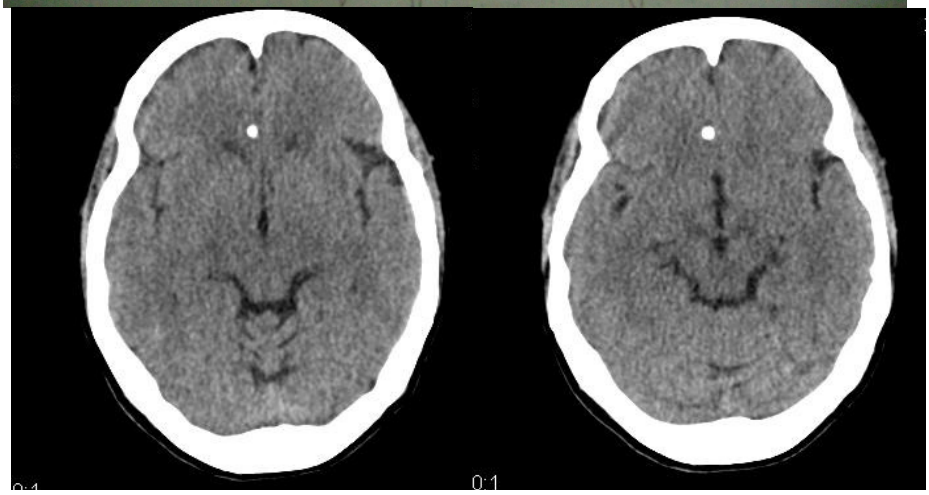
I



20 year old primigravida with GTCS type of seizure and disease free interval of 12 years. EEG was abnormal and CT showed calcified granuloma. She was on T. Phenytoin 2 HS. She delivered a baby with IUGR. BW was 2.5 Kg with Good Apgar score.

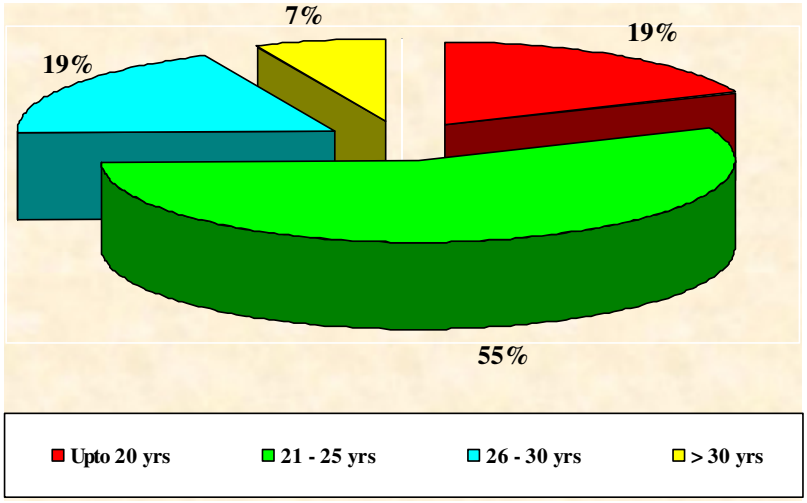
Case 4:





30 year old multigravida (G3 P2 L1) with last fit 6 year back on irregular intake of T. carbamazepine 1 tds. Her EEG was abnormal and CT shows calcified granuloma. LSCS done for previous LSCS with oligohydramnios. Baby birth weight was 2.5kg with normal Apgar.

AGE DISTRIBUTION



RESULTS AND ANALYSIS

Table 1: Age Distribution

Age (In years)	**Cases**	
	No	**%**
Up to 20 years	14	18.7
21-25 years	42	56
26-30 years	14	18.7
> 30 years	5	6.7
Total	75	100
Range	18-33 years	
Mean	24.1 years	
S.D	3.6 years	

* Majority of patient 42/ 75 (56%) belong to 21- 25 years of age group with mean age of 24.1 years

ANTENATAL CARE

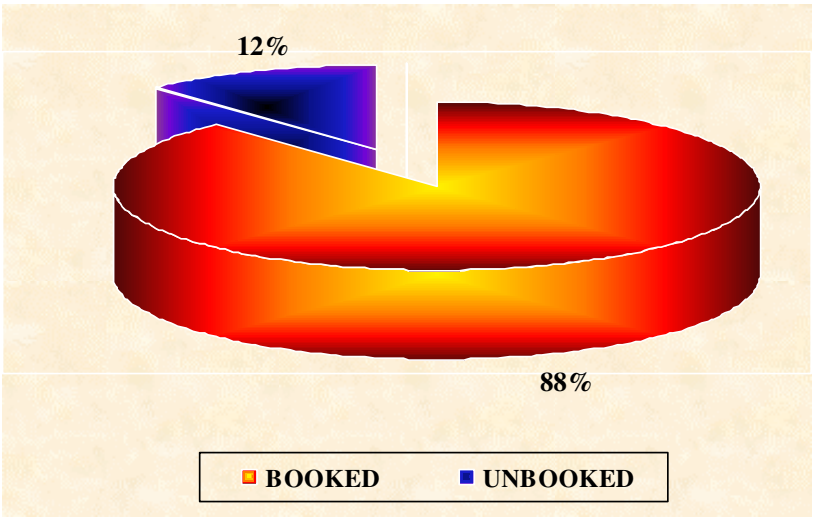


Table 2 : Antenatal Care

Antenatal Care	Cases	
	No	**%**
Booked	66	88
Unbooked	9	12
Total	75	100

* Booked cases was 66/75 (88 %) and 9/ 75 (12%) of cases were unbooked

PARITY

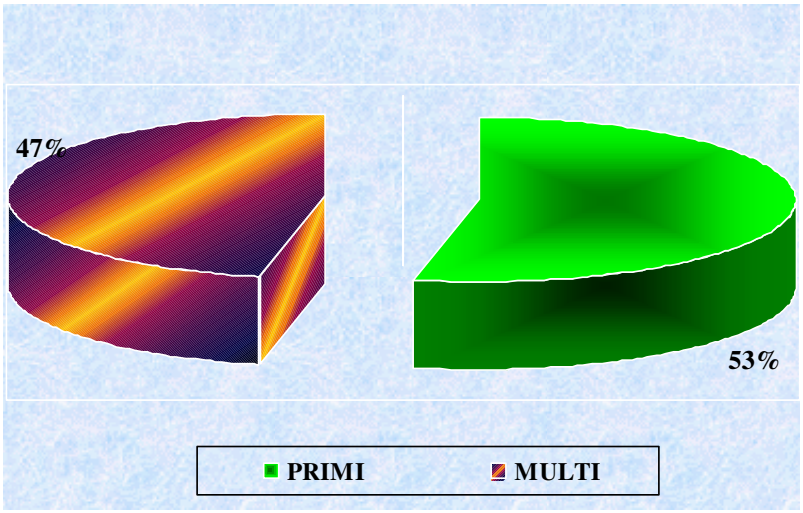


Table 3 : Parity

Parity	Cases	
	No	**%**
Primi	40	53.3
Multi	35	46.7
Total	75	100

- Out of 75 cases studied primigravida constitute 40 cases (53.3 %) and multigravida constitute 35 cases 46.7 %

DURATION OF DISEASE

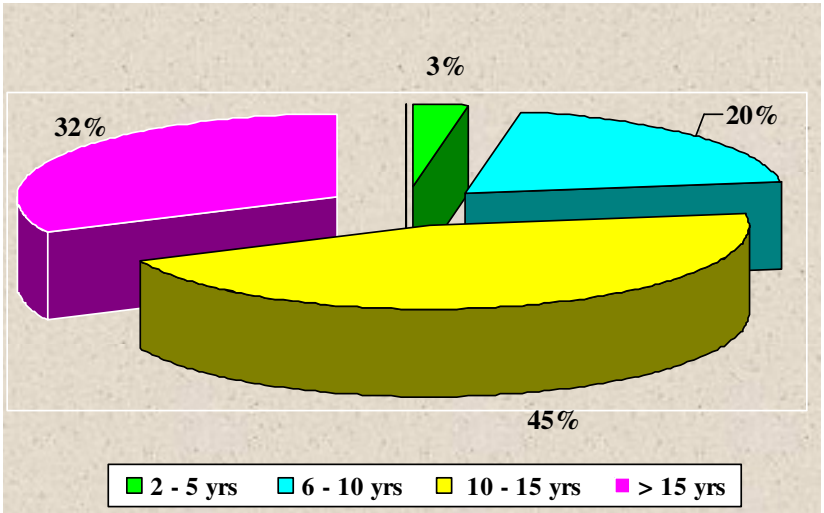


Table 4 : Duration of disease

Duration of disease (in years)	Cases	
	No	%
0-1 years	-	-
2 – 5 years	2	2.7
6- 10 years	15	20
10 – 15 years	34	45.3
> 15 years	21	32
Total	75	100
Range	3 – 25 years	
Mean	13.4 years	
S.D	4.1 years	

- 45.3 % of cases had epilepsy for duration for 10 -15 years. 77% of patients had duration of disease more than 10 years . none of the patient had duration of disease of less than 1 year.

Table 5 : Type of seizure

Type of seizure	Cases	
	No	**%**
GTCS	74	98.7
Partial	1	1.3
Total	75	100

* Out of 75 cases, 74 (98.7 %) had GTCS and only one case (1.3 %) had partial seizure

DISEASE FREE INTERVAL

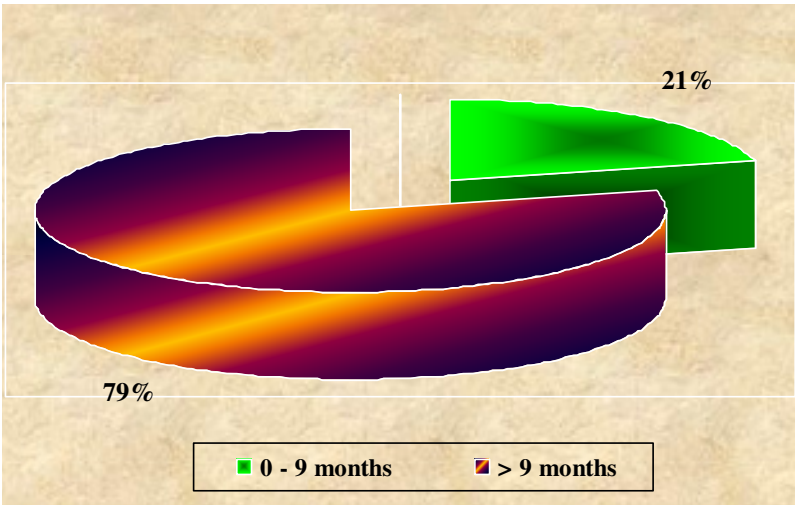


Table 6: Disease free interval

Disease free interval	Cases	
	No	**%**
0 – 9 months	16	21.3
> 9 months	59	78.7
Total	75	100
Range	2 days – 14 years	
Mean	5.1 years	
S.D	4.0 years	

* 16 cases out 75 (21.3%) had epileptic seizures during their antenatal period

TYPE OF AED THERAPY

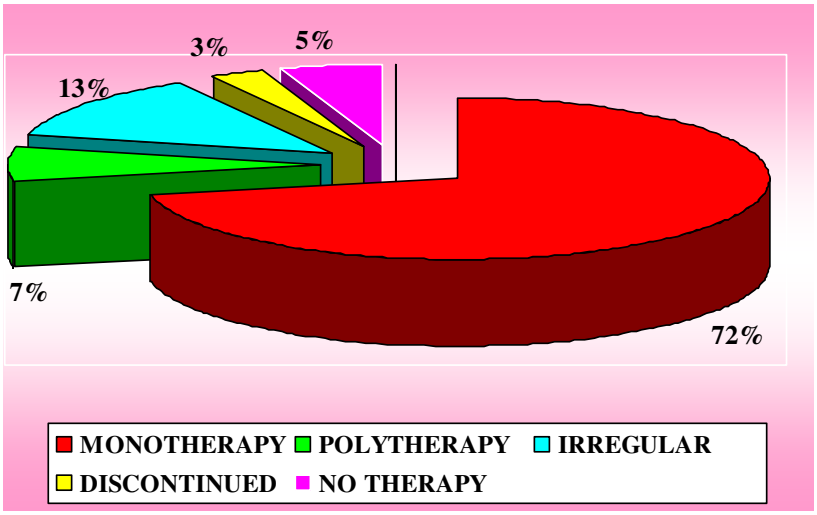


Table 7 : Type of AED therapy

Type of therapy	Cases	
	No	%
Mono therapy	54	72
Polytherapy	5	6.7
Irregular	10	13.3
Discontinued	2	2.7
Nil	4	5.3
Total	75	100

* Out of 75 cases studied of 54 cases (72%) were on monotherapy and 5 cases(6.7%) were on polytherapy. 13.3% had irregular intake of AED and 2.7% of cases discontinued the treatment after becoming pregnant. About 5.3% cases were not on any treatment as per neurologist advice.

FOLIC ACID INTAKE

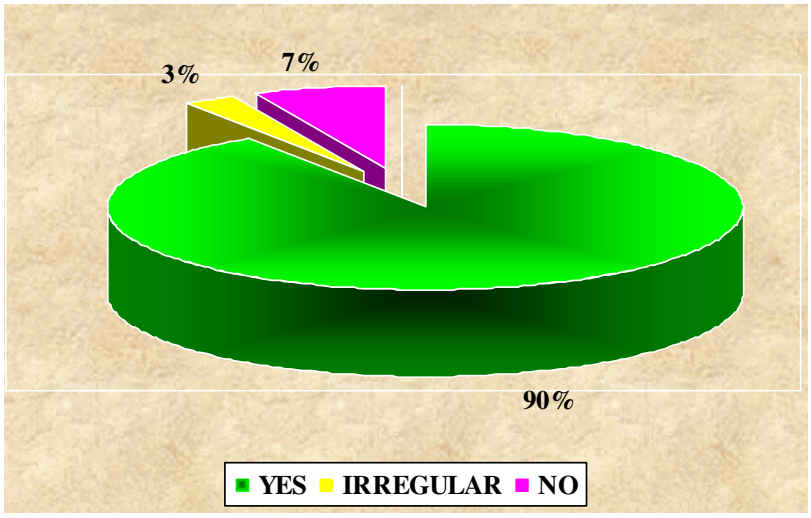


Table 8 : Folic acid intake

Folic acid intake	Cases	
	No	**%**
Yes	68	90.7
Irregular	2	2.7
No	5	6.7
Total	75	100

- 68/ 75 (90.7 %) of epileptic women with pregnancy were on folic acid along with AED.
- 2 women (2.7%) had irregular intake of folic acid and 5 women (6.7%) had no intake of folic acid.

EEG/ CT RESULTS

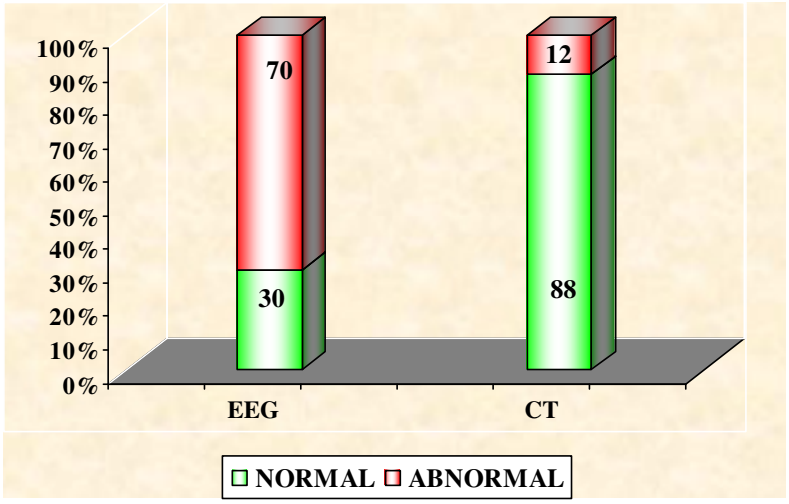


Table 9 : EEG / CT Abnormalities

Investigation	Results			
	Normal		Abnormal	
	No	%	No	%
EEG	23	30.7	52	69.3
CT	66	88	9	12

* Most of the women with epilepsy had abnormal EEG (69.3%) and normal CT (88%)

EEG was normal in 30.7% of cases while 12% had abnormal CT.

GESTATIONAL AGE AT DELIVERY

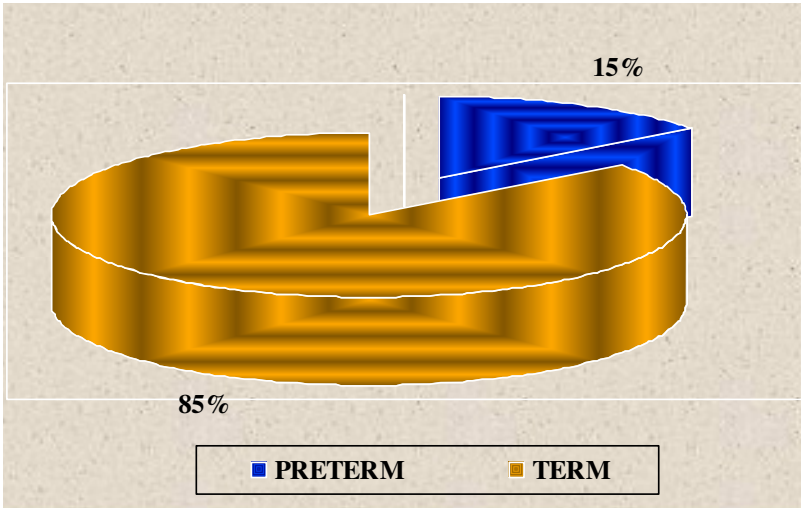


Table 10 : Gestational Age at delivery

Gestational Age at delivery (in weeks)	Cases	
	No	**%**
< 34 weeks	2	2.7
34 – 36 weeks	9	12
≥ 37 weeks	64	85.3
Preterm	11	14.7
Term	64	85.3
Total	75	100
Range	32 – 42 weeks	
Mean	37.4 weeks	
S.D	1.9 weeks	

* Gestational age at delivery ranged from 32 – 42 weeks in our study 11/75 cases (14.7%) had preterm delivery and 64/75cases (85.3 %) delivered at term.

MODE OF DELIVERY

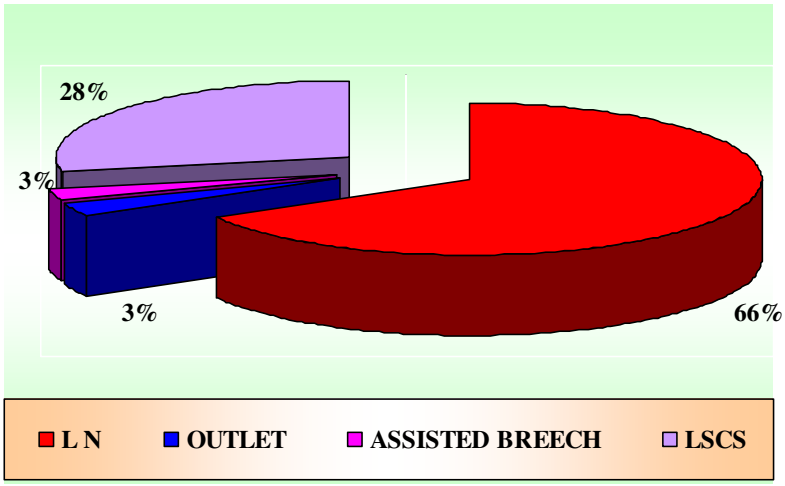


Table 11 : Mode of delivery

Mode of delivery	Cases	
	No	**%**
Labour natural	50	66.7
Outlet	2	2.7
Assisted breech	2	2.7
LSCS	21	28
Total	75	100

* 50/ 75 (66.7 %) of cases had normal delivery while 21/ 75 (28%)of cases underwent LSCS for various indication. Assisted breech and outlet forceps delivery accounted for 2.7% each.

BIRTHWEIGHT

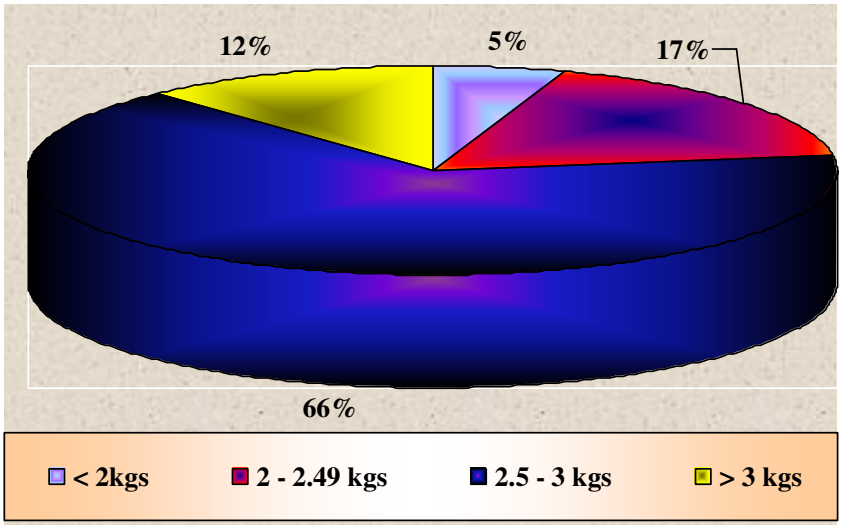


Table 12 : Birth Weight

Birth Weight (in kgs)	Cases	
	No	**%**
< 2 kgs	4	5.3
2 - 2.49 kgs	13	17
2.5 – 3 kgs	49	66
> 3 kgs	9	12
Total	75	100
Low birth weight (<2.5 kgs)	17	22.3
Term low birth weight	6	9.3
Range	1.1 – 3.8 kgs	
Mean	2.65 kgs	
S.D	0.44 kgs	

* 49/75 (66 %) of cases were of birth weight between 2.5 – 3 kg. BW of less than 2.5 kg accounted for17/ 75 (22.3%). Out of which term low birth weight constitute 6/75 (9.3%).

APGARSCORE

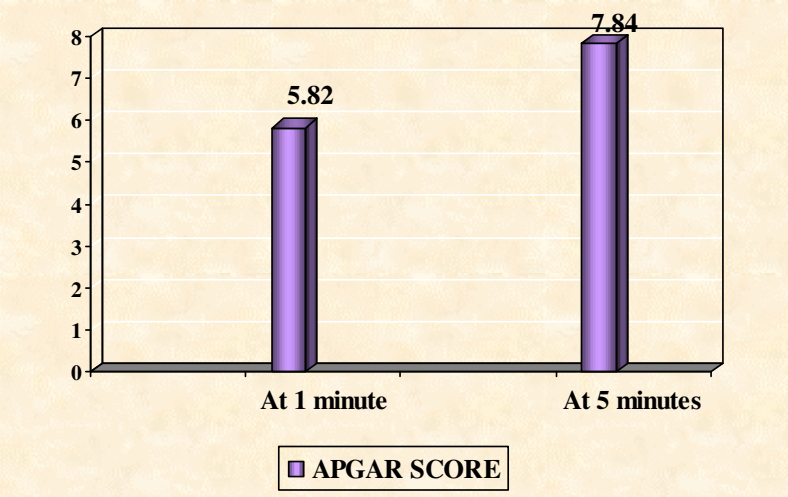


Table 13 : Apgar Score

Apgar Score	At 1 minute		At 5 minutes	
	No	%	No	%
2	2	2.7	-	-
3	1	1.4	-	-
4	1	1.4	1	1.4
5	-	-	1	1.4
6	70	94.6	2	2.7
7	-	-	1	1.4
8	-	-	69	93.2
Total	74•	100	74•	100
Range	2-6		4-8	
Mean	5.82		7.84	
S.D	0.77		0.66	

• One death

* 70/75 (94.6%) of the babies had good 1 min Apgar and 69/75 (93.2%) of the babies had good 5 min Apgar score.

Table 14 : Head Circumference

Parameter	Head Circumference (in cms)
Range	30-35 cms
Mean	33.5 cms
S.D	1.1 cms

* Average head circumference was 33.5 cms, with range of 30-35cms which was within normal limits.

OBSTETRIC COMPLICATIONS

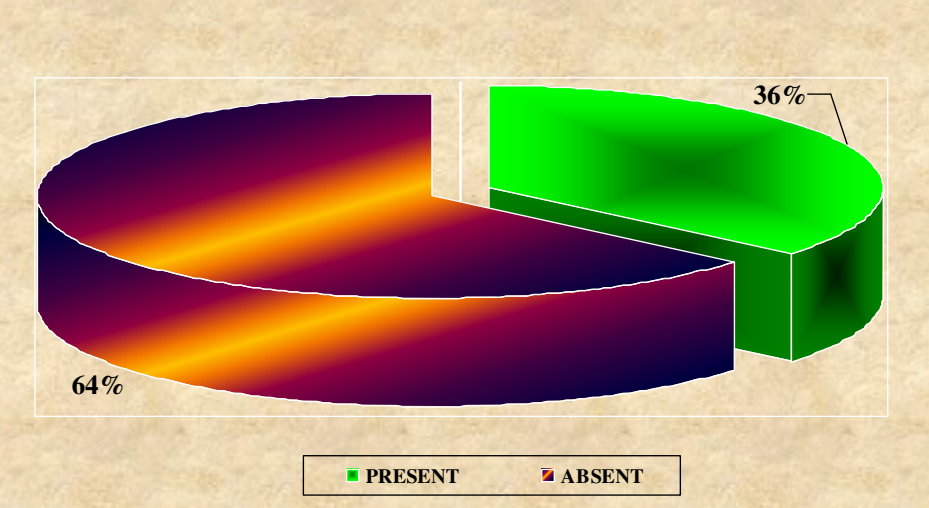


Table 15 : Obstetric Complications

Obstetric Complications	Cases	
	No	**%**
IUD with Preterm	1	1.3
IUGR	2	2.7
Malpresentation	4	5.3
Oligohydroamnios	2	2.7
Oligo & IUGR	2	2.7
Postdatism	6	8.0
Pre term	8	11
Preterm & Malpresentation	2	2.7
Total cases with complications	**27**	**36**
Cases without Complications	**48**	**64**
Total	**75**	**100**

* Out of 75 cases studied 48 cases (64%) had no complications and 27 cases (36%) had mentionable complications.

FREQUENCY OF SEIZURE

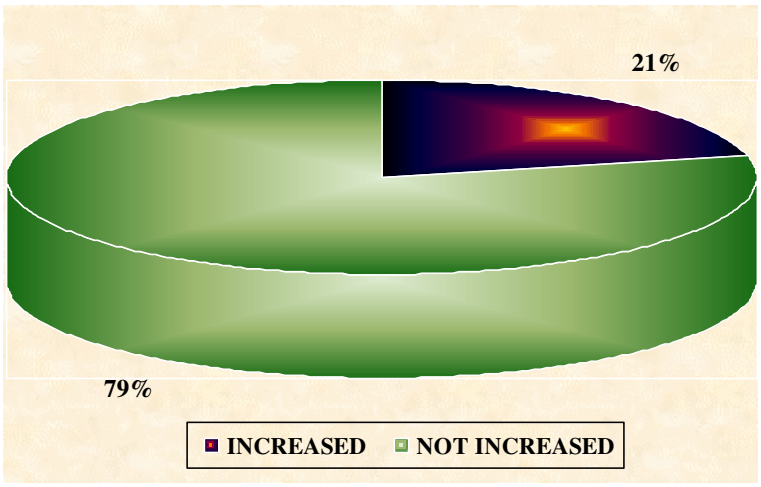


Table 16 : Frequency of seizures

Frequency of seizures	Cases	
	No	**%**
Increased	16	21.3
Not increased	59	78.7
Total	75	100

* Increase in frequency of seizures observed in 16/ 75 (21.3 %) of cases and there was no increase in 59/75 (78.7%) cases.

Table 17 : Maternal Outcome

Maternal Outcome	Cases	
	No	**%**
Good	75	100
Poor	Nil	-
Total	75	100

- Maternal outcome was good in all cases (100%).

Table 18 : Relationship between disease free interval and obstetric complications

Disease free interval	Obst. Complications			
	Present		Absent	
	No	%	No	%
0-1 years	7	33.3	14	66.7
> 1 year	14	25.9	40	74.1
Mean interval	5.77 years		4.84 years	
S.D	4.4 years		3.92 years	
'p'	0.3911 Not Significant			

* There is no statistically significant correlation between the disease free interval and occurrence of obstetric complications.

DISEASE FREE INTERVAL & FREQ. OF SEIZURES

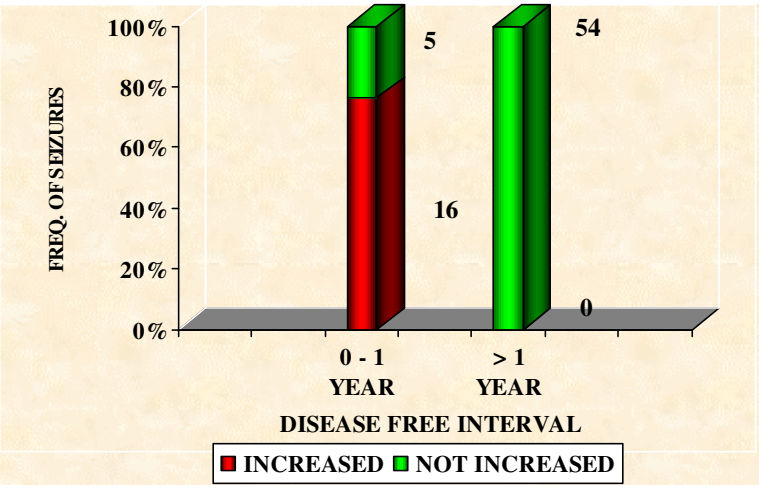


Table 19 : Relationship between disease free interval and frequency of seizures

Disease free interval	Frequency of seizures			
	Increased		Not increased	
	No	%	No	%
0-1 years	16	76.2	5	23.8
> 1 year	-	-	54	100
Mean interval (in yrs)	0.29 years		6.41 years	
S.D	0.27 years		3.57 years	
'p'	**0.0001** **Significant**			

* There is statistically significant correlation between the disease free interval and frequency of seizures.

DISCUSSION

Epilepsy is the second common chronic neurological disorder complicating pregnancy. Pregnancy in a mother with epilepsy brings about several concerns including the risk of recurrent seizures, seizure aggravation, increased obstetric risks and increased adverse neonatal outcome.

Review of literature shows vast majority of cases are uncomplicated but there are increased obstetric risks and increased adverse neonatal outcome when compared to general population. We made a prospective study comprising **75** women with epilepsy complicating pregnancy to assess the maternal and fetal outcome and occurrence of seizure frequency in mother.

In our study the age of the patients ranged from 18-33 years and mean maternal age was 24.1 years. The age group in our study is nearly comparable with the study conducted by (Thomas SV et al., 2001)[16] in which the maternal age range was from 19-38 years with mean age of 26.

In our study 53.3% of cases were primigravida and 46.7% of cases were multigravida whereas in the study by (Goel et al., 2006)[9] the majority of the case was of multigravida 70.2%. **Most of the cases in our study are booked cases (88%).**

The duration of epilepsy for more than 10 years accounted for 77% of cases in our study while in the study conducted by (Goel et al., 2006)[9], 72 % of the cases had duration of epilepsy for less than 10 years. **There were no cases with duration of epilepsy for less than 1 year in our study.**

Regarding the types of seizures, (Thomas et al., 2001)[16] observed, 59.38% had GTCS and 40.37% of other types. (Chattopadhyay et al., 2008)[5] in his study observed 56 % of GTCS and 44 % of other types. **In our study majority of the cases were of GTCS type (98.7%) and only one case (1.3%) had partial seizures.**

In our study 54 cases (72%) of patients were on monotherapy with 46 cases (61%) were on carbamazepine and 8 cases (10.6%) were on phenytoin. 6.7% of the patients were on polytherapy (combination of carbamazepine and phenytoin- 3 cases , carbamazepine and clobazam – 7 cases , phenytoin and lamotrigine – 1 case**) , 13.3% were on irregular treatment and 2 cases (2.7 %) discontinued AED after becoming pregnant.** In the study conducted by (Thomas SV et al., 2001)[16] 87.5% of cases were on monotherapy and 12% of cases were on polytherapy.

About 90.7% of patients on epileptic drug were on regular intake of folic acid and 2.7 % were on irregular intake, **while 6.7 % did not have any drug intake in our study.** According to (Thomas SV et al., 2001)[16] regular consumption of folic acid was documented in only in 40% of patients. **In our study majority of cases were on regular folic acid intake.**

Premature delivery had been reported by (Chattopadhyay et al., 2008) [5] for 18.6% and (Goel et al., 2006)[9] for 21.6% among their patients. **In our study 14.7% delivered prematurely and 85.3 % cases delivered at term.** It shows there is slight increase incidence of prematurity in epileptic women.

In our study, 66.7 % of cases had normal delivery while 28% of cases underwent LSCS for various indications including failed induction, fetal distress, oligohydroamnios, IUGR and postcaesarean pregnancy with associated complications. Assisted breech and outlet forceps delivery accounted for 2.7% each. Mode of delivery in epileptic women is nearly comparable to study of (Goel et al., 2006)[9], who observed normal vaginal delivery in 62% of cases and LSCS in 27% and instrumental delivery of 10.8%.

While (Thomas SV et.al 2001) [16] and (Chattopadhyay et al., 2008)[5] in their studies had more number of women undergoing LSCS accounting for 40.6% and 65.1% respectively. (Thomas SV et al., 2001)[16] had mentioned fetal distress, failed induction and uterine inertia as indication for LSCS.

Birth weight of neonates range from 1.1 -3.8 kg with mean birth weight of 2.65 kg. in our study. 5.5% of our babies had birth asphyxia (Apgar 1'= 2-6, 5'= 3-7). In (Thomas SV et al., 2001)[16] study the birth weight ranges from 1.8- 4.0 kg with mean weight of 2.84 kg. 10.7% of babies had birth asphyxia (Apgar 1' = 2-6, 5'= 3-7).

Birth weight of less than 2.5 kg accounted for 22.3% in our study. Similarly (Chattopadhyay et al., 2008)[5] has reported 23.3% and (Goel et al., 2006)[9] has reported 18% of low birth weight babies. (Thomas SV et al., 2001)[16] has reported 21 % of term low birth weight babies. **In our study term low birth weight constitute only 9.3%.**

Average head circumference was 33.5 cms, with range of 30-35cms which was within normal limits in our study. It was similar to (Thomas SV et al., 2001) [16] study where there was average head circumference of 35 cms, with range of 32- 44cms. There was no cases of microcephaly in our study.

Except for one fetus with congenital diaphragmatic hernia, whose mother was on phenytoin, there was no other congenital anomalies like neural tube, craniofacial, digital, cardiac/ urogenital defects in fetus of our epileptic women. (Thomas SV., 2011) [17] has mentioned in his study that GIT anomalies like esophageal atresia, omphalocele, hernia (diaphragmatic, inguinal and umbilical) can occur in women with AED.

In our study out of 75 cases, there were (5.3%) 4 cases of IUGR, (5.3%) 4 cases of oligohydramnios, (8%) 6 cases of malpresentation, (8%) 6 cases of postdatism. Preterm cases accounted for 14.7% (11 cases). There was one case of intrauterine death. Induction of labour was done in 14 cases (18.7%).

(Goel et al., 2006) [9] has reported PIH (24.3%), abruption (5.4%), GDM (2.7%) and induction of labour in (18.9) % of cases in his study. **Induction of labour was similar in our study, but there was no case of abruptio placenta, PIH or gestational diabetes.**

(Thomas SV et al., 2001)[16] observed over quarter of patients with PIH, preeclampsia, IUGR, postdatism, placental previa and hydraminos. (Chattopadhyay et al., 2008) [5] in observed IUGR (9.3 %) and preterm labour in (9.3%) of cases and there was no case of abruptio placenta, placenta previa, PIH or gestational diabetes. Type and occurrence of obstetric complications appear variable in different studies.

Seizure frequency was increased in 21.3% of our cases and not increased in 78.7% of cases. (Thomas et al., 2001)[16] in his study noted an increase in seizure frequency in 12.5% and no change in 78.1% of cases which is in accordance with our study.

One important outcome noted in our study is, there exist statistically significant correlation between the disease free interval and frequency of seizures, with a 'p' value of 0.0001 (Table 19). There was increased seizure frequency in 76.2% of cases with disease free interval of less than one year and there was no increase in seizure occurrence in which the disease free interval is more than one year.

There is no statistically significant correlation between the disease free interval and occurrence of obstetric complications (P > 0.05) (Table 18).

SUMMARY

- 75 epileptic women with pregnancy were assessed for maternal and fetal outcome including outcome of seizure frequency.
- Mean maternal age was 24.1 years.
- 53.3% were primigravida and 46.7% were multigravida.
- 77% had epilepsy for more than 10 years.
- 98.7% of patients had generalized tonic clonic seizures and 1.3% had partial seizures.
- 72 % of patients were on monotherapy and 6.7% of patients were on polytherapy.
- 90.7% of patients on antiepileptic drugs had regular intake of folic acid.
- 85.3% had term deliveries and 14.7% had preterm deliveries.
- 66.7% had normal delivery and 28% underwent caesarean sections for various obstetric indication.
- Low birth weight babies (< 2.5 kg) were seen in23.3% of patients. No cases of microcephaly were observed in our study and all babies had normal head circumference.
- Incidence of congenital anomalies was almost nil, except for one fetus with congenital diaphragmatic hernia.
- None of the patients had any significant obstetric complications.
- Seizure frequency was increased in 21.3%.

- There was a significant correlation between disease free interval and increase in seizure frequency. Shorter the disease interval higher the incidence of seizure frequency. 76.2 % of cases with disease free interval of less than one year had increased seizure frequency.

CONCLUSION

The maternal and fetal outcome was good in our study among the pregnant women with epilepsy, except for few complications like prematurity and low birth weight which has slightly increased.

The good maternal and fetal outcome with reduced occurrence of obstetric complications and reduced incidence of congenital anomalies in our study may be due to early booking, regular antenatal care, regular intake of anti epileptic drugs (preferentially monotherapy) and regular intake of folic acid along with antiepileptic drugs, which was found in most of our cases.

There is increased frequency of seizures in one fifth of our pregnant epileptic women with statistically significant correlation between the disease free interval and frequency of seizures. Shorter the disease interval higher the incidence of seizure frequency. Increased seizure frequency should be anticipated in patients with shorter disease free interval and may be advised to have regular antiepileptic drug intake and more frequent antenatal visits.

There exists no statistically significant correlation between the disease free interval and occurrence of obstetric complications.

Pregnant patients with epilepsy has to be considered as high risk pregnancy which need evaluation, specialist opinion and referral to tertiary care centres for better maternal and neonatal outcome. The maternal and fetal complications can be minimized by the close coordination between neurologist, obstetrician and the pediatrician.

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PROFORMA

NAME: AGE: IP No: Obstetric Code:

ADDRESS Booked / Unbooked LMP:

DOA: DOD: EDD:

COMPLAINTS:

Seizures -

Labour pain -

Draining P/V -

Bleeding P/V -

Fetal movements -

FH -

Edema legs -

Headache -

Nausea -

Vomiting -

Epigastric pain -

Oliguria -

PAST HISTORY:

SEIZURES:

* Age of onset -

* Type - GTCS / PARTIAL

* Disease free interval -

ANTIEPILEPTIC DRUG

* Drug and dose :

* Duration of intake :

* Regular / irregular :

FOLIC ACID INTAKE - Y / N

INVESTIGATIONS - EEG / CT

Status epilepticus -

Previous child drug effect -

HT / DM / PIH / TB / Renal disease

EXAMINATIONS:

Consciousness: C / SC / UC	**P/A** : Fundal Height -
Obeying commands: Y / N	I Pelvic grip -
Temperature: F / AF	FH -
Anemia: Y / N	
Edema legs: Y / N	
Pulse: / min BP: mm/hg	**P/V**: Cervix effacement -
CVS:	Dilatation -
RS:	Position -
CNS: EOM	Membrane -
PERL	Pelvis -
Limb movements	
DTR / Plantar reflex	
Meningeal signs	
Optic Fundus:	
Neuro Opinion:	

INVESTIGATIONS:

Blood Hb % -	LFT :
Urine Alb -	Blood Platelet count -
Sugar -	
Deposit -	USG - GA / AFI/ ANOMALIES
Blood: Urea -	
Sugar -	EEG: N / Ab
Serum: Creatinine -	CT : N / Ab
Uric acid -	
Serum electrolytes -	

LABOUR:

Spontaneous / Induced -

Mode of Delivery: Vaginal / Instrumental / LSCS

Indication:

Complications:

FETAL OUTCOME

Birth weight -

Apgar score -

Anomalies -

Head circumference -

IUD / Still Birth –

MASTER CHART

No	Age(yrs)	IP No	Booked	Obs code	Seizures			Drugs intake		Investigations		Delivery		Fetal outcome				Maternal outcome			
					Age onset	Type	Free interval yr	Anti Epileptic therapy	Folic Acid	EEG	CT	GA (weeks)	Mode	BW kg	APGAR 1'	APGAR 5'	HC(cms)	ANOM	Obst. Compli.	SZ . freq	Outcome
1	25	4722	YES	PRIMI	13	GTCS	11	POLY	YES	AB	NORMAL	36	LN	2.4	6	8	33	Nil	Preterm	No increase	GOOD
2	26	4893	YES	MULTI	7	GTCS	0.5	MONO	YES	AB	NORMAL	37	LSCS	3.5	6	8	33	Nil	Mal.pres	Increase	GOOD
3	21	48424	YES	MULTI	9	GTCS	10	MONO	YES	N	NORMAL	38	LSCS	2.5	6	8	34	Nil	Oligo	No increase	GOOD
4	20	48158	NO	MULTI	7	GTCS	13	MONO	YES	N	NORMAL	38	LN	2.7	6	8	33	Nil	Nil	No increase	GOOD
5	25	48396	YES	PRIMI	12	GTCS	13	IRREG.	YES	N	NORMAL	41	LSCS	2.3	6	8	33	Nil	Post datism	No increase	GOOD
6	25	9998	YES	PRIMI	13	GTCS	0.17	MONO	YES	N	NORMAL	38	LSCS	3	6	8	33	Nil	Nil	Increase	GOOD
7	23	9995	YES	MULTI	20	GTCS	0.4	POLY	YES	AB	CG	38	LSCS	2.8	6	8	34	Nil	Nil	Increase	GOOD
8	23	9032	YES	MULTI	12	GTCS	12	MONO	YES	N	NORMAL	37	LN	2.5	6	8	33	Nil	Nil	No increase	GOOD
9	23	9690	NO	PRIMI	16	GTCS	0.33	IRREG.	IRREG.	AB	NORMAL	38	LN	2.3	6	8	34	Nil	Nil	Increase	GOOD
10	23	9135	YES	MULTI	12	GTCS	11	MONO	YES	N	NORMAL	37	LSCS	3	6	7	33	Nil	Mal.pres	No increase	GOOD
11	19	8499	YES	PRIMI	9	GTCS	0.5	MONO	YES	AB	NORMAL	38	LN	2.8	6	8	33	Nil	Nil	Increase	GOOD
12	21	9361	YES	PRIMI	5	GTCS	7	MONO	YES	AB	NORMAL	41	LSCS	3	2	6	34	Nil	Post datism	No increase	GOOD
13	20	9418	YES	PRIMI	6	GTCS	7	MONO	YES	AB	CG	39	LN	2.8	6	8	33	Nil	Nil	No increase	GOOD
14	25	8628	YES	MULTI	10	GTCS	0.5	IRREG.	YES	AB	NORMAL	38	Outlet	2.5	6	8	33	Nil	Nil	Increase	GOOD
15	21	8286	YES	PRIMI	9	GTCS	12	MONO	YES	AB	CG	37	LN	2.5	6	8	34	Nil	Nil	No increase	GOOD
16	32	8243	YES	PRIMI	20	GTCS	4	MONO	YES	AB	NORMAL	38	LN	3	6	8	33	Nil	Nil	No increase	GOOD
17	33	7053	YES	PRIMI	13	GTCS	14	IRREG.	IRREG	AB	NORMAL	38	LN	2.5	6	8	34	Nil	Nil	No increase	GOOD
18	22	6629	YES	MULTI	9	GTCS	3	POLY	YES	N	NORMAL	38	LSCS	2.5	6	8	35	Nil	Nil	No increase	GOOD
19	25	7402	YES	PRIMI	15	GTCS	0.009	IRREG.	YES	AB	NORMAL	38	LSCS	2.2	6	8	33	Nil	Oligo, IUGR	Increase	GOOD

No	Age(yrs)	IP No	Booked	Obs code	Seizures			Drugs intake		Investigations		Delivery		Fetal outcome				Maternal outcome			
					Age onset	Type	Free interval yr	Anti Epileptic therapy	Folic Acid	EEG	CT	GA (weeks)	Mode	BW kg	APGAR 1'	APGAR 5'	HC(cms)	ANOM	Obst. Compli.	SZ . freq	Outcome
20	18	11491	YES	PRIMI	15	GTCS	3	MONO	YES	N	NORMAL	38	LSCS	2.3	6	8	33	Nil	Nil	No increase	GOOD
21	29	10081	YES	MULTI	13	GTCS	0.67	MONO	YES	AB	NORMAL	34	LN	2.2	6	8	32	Nil	PRE TERM	Increase	GOOD
22	30	1841	NO	MULTI	14	GTCS	0.006	NIL	YES	AB	NORMAL	38	LSCS	2.7	6	8	34	Nil	Mal.pres	Increase	GOOD
23	26	1796	YES	MULTI	8	GTCS	0.33	POLY	YES	AB	NORMAL	37	LN	2.7	6	8	35	Nil	Nil	Increase	GOOD
24	21	1023	YES	MULTI	7	GTCS	4	MONO	YES	AB	NORMAL	35	ABD	2.4	6	8	35	Nil	PRE TERM Mal.pres	No increase	GOOD
25	25	1338	NO	MULTI	9	GTCS	11	MONO	YES	N	NORMAL	39	LN	2	6	8	33	Nil	IUGR	No increase	GOOD
26	19	10518	YES	PRIMI	10	GTCS	0.33	MONO	YES	AB	NORMAL	38	LN	3.2	6	8	34	Nil	Nil	Increase	GOOD
27	28	10547	YES	MULTI	18	GTCS	6	IRREG.	YES	AB	CG	37	LSCS	2.5	6	8	33	Nil	OLIGO	No increase	GOOD
28	23	1165	YES	MULTI	14	GTCS	0.6	MONO	YES	AB	NORMAL	38	LSCS	3	6	8	35	Nil	Nil	Increase	GOOD
29	21	1090	YES	PRIMI	13	GTCS	3	MONO	YES	AB	NORMAL	36	LN	2.3	2	4	33	Cong.diaph.hernia	PRE TERM	No increase	GOOD
30	25	2970	YES	PRIMI	11	GTCS	10	MONO	YES	N	NORMAL	37	LSCS	3.1	6	8	35	Nil	Nil	No increase	GOOD
31	23	2403	YES	MULTI	11	GTCS	6	MONO	YES	AB	CG	39	LSCS	2.5	6	8	33	Nil	Nil	No increase	GOOD
32	26	2935	YES	PRIMI	10	GTCS	3	MONO	YES	N	NORMAL	37	LN	3.3	6	8	34	Nil	Nil	No increase	GOOD
33	24	3149	YES	MULTI	9	GTCS	3	MONO	YES	AB	NORMAL	38	LSCS	2.7	6	8	33	Nil	Nil	No increase	GOOD
34	25	57942	YES	MULTI	7	GTCS	2	MONO	YES	AB	NORMAL	37	LN	2.8	6	8	35	Nil	Nil	No increase	GOOD
35	20	57508	YES	PRIMI	12	GTCS	11	MONO	YES	AB	NORMAL	37	LN	2.5	6	8	33	Nil	Nil	No increase	GOOD
36	25	66069	YES	PRIMI	11	GTCS	9	MONO	YES	AB	NORMAL	34	LN	2.1	6	8	32	Nil	PRE TERM	No increase	GOOD
37	20	6073	NO	PRIMI	10	GTCS	9	IRREG.	YES	N	NORMAL	34	LN	2	6	8	31	Nil	PRE TERM	No increase	GOOD
38	23	61299	YES	MULTI	7	GTCS	0.17	MONO	YES	AB	CG	37	LN	3.8	6	8	33	Nil	Nil	Increase	GOOD
39	25	9242	NO	MULTI	8	GTCS	1	MONO	YES	AB	NORMAL	34	LN	1.8	Dead	Dead	Dead	Nil	PRE TERM	No increase	GOOD

No	Age(yrs)	IP No	Booked	Obs code	Seizures			Drugs intake		Investigations		Delivery		Fetal outcome				Maternal outcome			
					Age onset	Type	Free interval yr	Anti Epileptic therapy	Folic Acid	EEG	CT	GA (weeks)	Mode	BW kg	APGAR 1'	APGAR 5'	HC(cms)	ANOM	Obst. Compli.	SZ . freq	Outcome
																			IUD		
40	32	59639	YES	MULTI	7	GTCS	3	MONO	YES	AB	NORMAL	34	LN	2	6	8	32	Nil	PRE TERM	No increase	GOOD
41	20	8016	YES	PRIMI	9	GTCS	6	MONO	YES	AB	CG	32	ABD	1.6	4	6	30	Nil	PRE TERM Mal.pres	No increase	GOOD
42	23	6362	YES	MULTI	9	GTCS	4	MONO	YES	AB	NORMAL	37	LN	2.5	6	8	33	Nil	Nil	No increase	GOOD
43	20	55177	YES	PRIMI	13	GTCS	7	IRREG.	YES	AB	NORMAL	37	LN	2.5	6	8	33	Nil	Nil	No increase	GOOD
44	23	5220	YES	MULTI	9	GTCS	6	MONO	YES	AB	NORMAL	37	LN	3	6	8	33	Nil	Nil	No increase	GOOD
45	29	5013	YES	MULTI	12	GTCS	9	MONO	YES	AB	NORMAL	37	LN	3	6	8	33	Nil	Nil	No increase	GOOD
46	20	1863	YES	PRIMI	7	GTCS	0.08	DISCONT.	NO	AB	NORMAL	38	LN	3.1	6	8	35	Nil	Nil	Increase	GOOD
47	23	7075	YES	MULTI	7	GTCS	6	MONO	YES	AB	NORMAL	39	LN	3	6	8	35	Nil	Nil	No increase	GOOD
48	23	2590	YES	MULTI	5	GTCS	0.006	MONO	YES	AB	NORMAL	38	LN	3.2	6	8	34	Nil	Nil	Increase	GOOD
49	29	1936	YES	MULTI	5	GTCS	5	MONO	YES	N	NORMAL	42	LN	2.7	6	8	33	Nil	Postdatism	No increase	GOOD
50	30	2098	YES	MULTI	18	GTCS	11	MONO	YES	AB	CG	37	LN	2.7	6	8	34	Nil	Nil	No increase	GOOD
51	28	3678	YES	MULTI	8	GTCS	8	MONO	YES	AB	NORMAL	38	LN	2.7	6	8	33	Nil	Nil	No increase	GOOD
52	21	2549	YES	PRIMI	9	GTCS	4	IRREG.	YES	AB	NORMAL	37	LN	2.8	6	8	35	Nil	Nil	No increase	GOOD
53	33	14151	YES	MULTI	13	GTCS	6	MONO	YES	N	NORMAL	38	LN	2.9	6	8	35	Nil	Nil	No increase	GOOD
54	21	12675	YES	PRIMI	12	GTCS	8	MONO	YES	AB	NORMAL	41	LN	2.7	6	8	34	Nil	Postdatism	No increase	GOOD
55	23	7810	YES	PRIMI	9	GTCS	2	MONO	YES	AB	NORMAL	37	LN	2.8	6	8	34	Nil	Nil	No increase	GOOD
56	23	7429	YES	MULTI	13	GTCS	3	MONO	YES	N	NORMAL	37	LN	2.7	6	8	35	Nil	Nil	No increase	GOOD
57	31	9548	YES	MULTI	12	GTCS	1	MONO	YES	AB	NORMAL	38	LSCS	2.8	6	8	34	Nil	Nil	No increase	GOOD
58	20	7989	NO	PRIMI	8	GTCS	8	MONO	YES	N	NORMAL	37	LN	2.7	6	8	34	Nil	Nil	No increase	GOOD
59	22	52829	YES	PRIMI	11	GTCS	3	IRREG.	YES	AB	NORMAL	41	LN	2.8	6	8	33	Nil	Postdatism	No increase	GOOD

No	Age(yrs)	IP No	Booked	Obs code	Seizures			Drugs intake		Investigations		Delivery		Fetal outcome				Maternal outcome			
					Age onset	Type	Free interval yr	Anti Epileptic therapy	Folic Acid	EEG	CT	GA (weeks)	Mode	BW kg	APGAR 1'	APGAR 5'	HC(cms)	ANOM	Obst. Compli.	SZ . freq	Outcome
60	26	10535	YES	PRIMI	12	GTCS	0.03	MONO	YES	AB	NORMAL	39	LN	2.8	6	8	34	Nil	Nil	Increase	GOOD
61	20	7985	YES	PRIMI	9	GTCS	9	MONO	YES	N	NORMAL	37	LSCS	2.7	6	8	34	Nil	OLIGO, IUGR	No increase	GOOD
62	19	9631	YES	PRIMI	7	GTCS	1	MONO	YES	N	NORMAL	34	LN	1.8	6	8	31	Nil	PRETERM	No increase	GOOD
63	25	6055	YES	PRIMI	8	GTCS	0.9	MONO	YES	AB	NORMAL	32	LN	1.1	3	5	30	Nil	PRETERM	No increase	GOOD
64	25	5384	YES	PRIMI	9	GTCS	9	NIL	NO	N	NORMAL	37	LSCS	3	6	8	34	Nil	Nil	No increase	GOOD
65	30	65225	YES	MULTI	13	GTCS	2	POLY	YES	AB	CG	39	LN	3.5	6	8	35	Nil	Nil	No increase	GOOD
66	24	7902	NO	PRIMI	12	GTCS	1	MONO	YES	N	NORMAL	38	LN	2.6	6	8	32	Nil	Nil	No increase	GOOD
67	28	47515	YES	PRIMI	8	GTCS	4	MONO	YES	N	NORMAL	41	Outlet	2.8	6	8	34	Nil	Postdatism	No increase	GOOD
68	25	47439	YES	PRIMI	12	GTCS	9	NIL	NO	N	NORMAL	37	LN	2.5	6	8	33	Nil	Nil	No increase	GOOD
69	25	46396	YES	PRIMI	13	GTCS	10	NIL	NO	AB	NORMAL	37	LSCS	2.3	6	8	33	Nil	IUGR	No increase	GOOD
70	21	48421	YES	MULTI	11	GTCS	7	MONO	YES	AB	NORMAL	37	LN	2.7	6	8	34	Nil	Nil	No increase	GOOD
71	23	5215	YES	MULTI	13	Partial	6	MONO	YES	AB	NORMAL	39	LN	2.8	6	8	33	Nil	Nil	No increase	GOOD
72	24	2978	NO	PRIMI	13	GTCS	5	MONO	YES	AB	NORMAL	37	LN	2.9	6	8	34	Nil	Nil	No increase	GOOD
73	20	91840	YES	PRIMI	12	GTCS	4	MONO	YES	AB	NORMAL	38	LN	2.7	6	8	33	Nil	Nil	No increase	GOOD
74	25	1400	YES	PRIMI	13	GTCS	6	MONO	YES	N	NORMAL	37	LN	2.6	6	8	34	Nil	Nil	No increase	GOOD
75	26	10571	YES	PRIMI	10	GTCS	6	DISCONT.	NO	AB	NORMAL	38	LSCS	3.5	6	8	35	Nil	Nil	No increase	GOOD