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Obstetric Medicine

NEWSLETTER

SOCIETY OF
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From the Editor's Desk

Dear SOMI Members,

Greetings from Hyderabad. This issue of SOMI e-newsletter focuses on some of the neurological disorders complicating pregnancy. This issue covers common problems encountered in pregnancy like headache and epilepsy to uncommon disorders like stroke complicating pregnancy. Epilepsy per se is not associated with congenital anomalies but anti-epileptic drugs have teratogenic potential. Despite this teratogenicity, anti-epileptic drugs need to be continued in pregnancy as seizures in pregnancy are associated with the worse fetal outcomes. It is important for women with epilepsy to have preconceptional counselling and have the drugs changed to ones with least teratogenic potential. Headache is a common problem in pregnancy. It is important to identify the etiology as it may indicate imminent eclampsia, cerebral venous thrombosis or a benign headache. Women with history of venous thromboembolism need anticoagulation during and antenatal period and postpartum period. Cerebrovascular accident can be thrombotic and hemorrhagic. Thrombolysis is not contraindicated in pregnancy and can be considered in severe cases.

Obstetricians and physicians need to be aware of common neurological disorders complicating pregnancy. CT scan of brain with an abdominal shield is safe in pregnancy. However, MRI is safe and more useful in detecting small infarcts and cerebral sinus venous thrombosis. Women with preexisting neurological disorders should be encouraged to attend preconception counselling clinic and a multidisciplinary team involvement is necessary for optimal maternal and fetal outcomes.

Regards,
Dr. Hari Kishan Boorugu
Editor

NEWSLETTER Obstetric Medicine

Anti-epileptic Drugs – Pharmacology & Teratogenecity

– Dr. Sruhya
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Introduction

Epilepsy in pregnancy:

The incidence of epilepsy in pregnant women is in between 0.3% and 0.6%. In women with epilepsy, seizure frequency in pregnancy remains unchanged in 54% to 80%, increases in 14% to 32% and decreases in 3% to 24%. Women with focal seizures have risk. Overall, seizure frequency in pregnancy mainly depends on the control pre-conceptionally i.e., in patients who were seizure free for at least 9 months before conception, the likelihood of remaining seizure free during pregnancy is 84 % to 92%.

Need for Anti-epileptic drugs:

Though the risk of congenital malformations for fetus in women with epilepsy on Anti-epileptic Drugs is double that of the general population, 90% have a good outcome. Risk is more with untreated epilepsy than Anti-epileptic drugs per se. Therefore, in women with epilepsy, Anti-epileptic drugs should not be stopped immediately after conception for the fear of teratogenicity.

Classification of Anti–epileptic Drugs:

Anti–epileptic drugs are classified in the following 3 ways.

1. Older and newer generation anti-epileptic drugs
2. Based on the Mechanism of Action
3. Based on Chemical Composition

Older and newer anti-epileptic drugs

Older Anti-epileptic Drugs	Newer Anti-epileptic Drugs
• Valproic acid	• Lamotrigine
• Phenytoin	• Levetiracetam
• Phenobarbitone	• Oxcarbazepine
• Carbamazepine	• Eslicarbazepine
• Clonazepam	• Topiramate
• Clobazam	• Tiagabine
• Ethosuximide	• Vigabatrine
	• Zonisamide
	• Lacosamide
	• Gabapentine
	• Felbamate
	• Pregabaline
	• Rufinamide

Anti-epileptic Drugs – Pharmacology & Teratogenecity

Based on the Mechanism of Action:

S. No.	Mechanism of action	Drugs
1.	Sodium channel blockers	Valproic acid Carbamazepine Phenytoin Lamotrigine
2.	Calcium channel blockers (t - type)	Ethosuximide Valproic acid
3.	Voltage gated calcium channel blockers	Phenytoin Gabapentine Pregabalin
4.	Glutamate blockers (NMDA)	Felbamate
5.	Glutamate blockers (ampa/kainate) Mechanism of action	Topiramate Drugs
6.	Carbonic anhydrase inhibitors	Topiramate Zonesamide
7.	Stereo selective binding site at synaptic plasma membrane	Levetiracetam
8.	GABA – A agonists	Phenobarbitone Clonazepam Clobazam
9.	Block presynaptic GABA reuptake	Tiagabine
10.	GABA transaminase inhibitor	Vigabatrin
11.	Glutamic acid decarboxylase modulator	Gabapentin Valproic acid

Based on Chemical Composition:

S. No.	Group	Drug
1.	Barbiturate	Phenobarbitone
2.	Deoxybarbiturate	Primidone
3.	Hydantoin	Phenytoin Fosphenytoin
4.	Iminostilbene	Carbamazepine Oxcarbazepine
5.	Succinimide	Ethosuximide
6.	Aliphatic carboxylic acid	Valproic acid Divalproex
7.	Benzodiazepines	Diazepam Lorazepam Clonazepam Clobazam
8.	Phenyltriazine	Lamotrigine
9.	Cyclic GABA analogue	Gabapentin

10.	Newer drugs	Vigabatrin Topiramate Tiagabine Zonisamide Levetiracetam

Brief Review of Individual Drugs:

1. Valproic acid:

It is metabolized by glucuronidation and beta – oxidation mainly in liver, its t ½ is 9 to 16 hours.

Uses: It is a broad spectrum anti-epileptic drug used in all types of epilepsy and also in migraine prophylaxis.

Dose: 10mg/kg/day. Max – 60mg/kg/day

Contraindications: Avoid in hepatic disease, urea cycle disorders, inborn errors of carnitine metabolism, cytopenias

Teratogenicity: It is 4 fold increased compared to other AEDs. It causes Fetal Valproate Syndrome which includes the following features:

- **Cranio-facial:** High forehead, Short broad nose, Long philtrum with thin vermilion border, Micrognathia, Cleft palate, Mid face hypoplasia
- **CVS:** Hypoplastic left heart, Aortic valve stenosis, Interrupted aortic arch, Secundum type ASD, VSD
- **CNS:** Spina bifida, Septo optic dysplasia
- **Extremities:** Long, thin fingers and toes, Post axial polydactyly, Radial ray reduction defects

1st trimester use of Sodium Valproate is associated with 7 fold increased risk of congenital malformations. According to EURAP study, when it is withdrawn in 1st trimester, the risk of seizure frequency was 32% compared to those who continued (16%). The frequency of malformations is dose dependent.

2. Carbamazepine:

It is an enzyme inducer that induces its own metabolism. Liver metabolism leads to an active metabolite. Inactivation occurs by hydroxylation and conjugation. Its t ½ is initially 25 to 65 hours and later 12 to 17 hours.

Uses: It is used in GTCS, Simple partial and complex partial seizures. Also used in Trigeminal neuralgia and Bipolar Mood Disorder.

Dose: Oral - 200 mg twice daily initially and maximum – 1600 mg / day.

Contraindications: Do not use in absent / atonic/ myoclonic seizures. Also contraindicated in some forms of porphyrias, cardiac arrhythmias, glaucoma.

Teratogenicity:

- **Fetal Carbamazepine Syndrome:** It includes Facial dysmorphism (upslanting palpebral fissures and long philtrum), developmental delay, spina bifida, distal phalanx and finger nail hypoplasia

3. Oxcarbazepine:

It is metabolized to an active form in liver. More than 95% is excreted through kidneys. Both the drug and its active form inhibits CYP 2C19 and stimulates CYP 3A4.

Uses: It is used in partial seizures

Dose: Oral – 300 mg twice daily and maximum dose is 2400 mg /day.

Contraindications: It is used cautiously in renal disease.

Adverse effects: It can cause hypernatremia, hypokalemia, hypocalcaemia, visual impairment, pancreatitis and hypothyroidism.

Teratogenicity: Can cause oral clefts and VSD.

4. Phenytoin:

It is metabolized in liver and is an enzyme inducer. Initially it follows 1st order kinetics and later 0 order kinetics.

Uses: It is used in GTCS, complex partial seizures and in neuropathic pain syndromes.

Dose: For GTCS / partial seizures – Loading dose – 15- 20 mg/kg oral and maintenance dose – 4 – 7 mg/ kg /day.

Contraindications: Do not use in Hydantoin hypersensitivity, liver disease, heart blocks

Adverse effects: Can cause hyperglycemia, gingival hyperplasia and decreased bone mineral density on chronic use.

Teratogenicity: Fetal Hydantoin Syndrome – It includes:

- **Cranio facial:** Hypertelorism, broad, depressed nasal bridge, cleft lip & palate, ptosis and microcephaly.
- **Limbs:** Distal phalanges hypoplasia, Digitalized thumb, Cone shaped epiphyses and Hip dislocation
- **CVS:** Aortic stenosis, Pulmonary stenosis and PDA
- **GIT:** Duodenal atresia and Anal atresia

5. Phenobarbitone:

It is long acting barbiturate with t ½ - 50 to 120 hours. About 75 % is metabolized in liver by CYP 2C9 with 25 % excreted unchanged in urine. It is an enzyme inducer.

Uses: Used in all types of seizures except absent seizures.

Dose: For status epilepticus 10 mg/kg /iv followed by 5 mg/kg.

Contraindications: Do not use in barbiturate, phenytoin and benzodiazepine hypersensitivity, COPD, severe hepatic disease. Use cautiously in renal impairment and porphyrias.

Monitor for hemorrhagic disease of new born in the neonate.

6. Ethosuximide

It is metabolized in liver by hydroxylation and 12% to 20% is excreted unchanged in urine.

Uses: Drug of choice for absent seizures.

Dose: Start with 250 mg twice daily and maximum is 1.5 gram / day.

Contraindications: Avoid in hepatic disease, porphyrias, bone marrow depression.

Teratogenicity: Can cause bilateral cleft lip.

7. Clonazepam:

It is mainly metabolized in liver with < 2% excreted unchanged in urine.

Uses: In absent seizures, akinetic and myoclonic seizures and in restless leg syndrome.

Dose: For seizures - 1.5 mg/ day oral and maximum dose in 20 mg /day.

Contraindications: In Benzodiazepine hyper sensitivity and acute angle closure glaucoma. Use cautiously in COPD, muscular dystrophy, myasthenia gravis and porphyria.

It may cause withdrawal effects in newborn.

8. Clobazam:

It mainly undergoes liver metabolism, weak inducer of CYP 3A4 and inhibitor of CYP 2D6.

Uses: Adjuvant treatment for Lennox Gestaut syndrome and in other epilepsies

Dose: Oral – 5 mg /day and maximum dose is 40 mg /day.

Contraindications: In hypersensitivity and avoid co-treatment with other CNS depressants especially opioids.

Teratogenicity: It can cause Floppy Infant Syndrome when given prior to delivery which lasts for 2 weeks. It includes hypothermia, respiratory depression, hypotonia and difficult feeding.

It can cause Neonatal Abstinence Syndrome on withdrawal. It includes hypertonia, hyperreflexia, hyperventilation, tremor, diarrhea and vomitings till 3 weeks.

Insufficient evidence on neurodevelopment.

9. Lamotrigine:

It is metabolized liver and 10% is excreted unchanged in urine. It is an enzyme inducer – induces its own metabolism.

Uses: In partial seizures, Bipolar Mood Disorder and Lennox – Gastaut Syndrome.

Dose: Start with 50 mg once daily and maintenance dose is 250 mg twice daily.

Contraindications: Avoid in hypersensitivity, hemphagocytic lymphohistiocytosis and aseptic meningitis.

Adverse effect: It can cause arthralgia, myalgia, SIADH.

Teratogenicity: In the International Lamotrigine Pregnancy Registry, they observed 1st trimester exposure to Lamotrigine and risk of Major Congenital

Malformations over a period of 18 years. They observed no increased risk with the monotherapy (2.2%), increased risk with Lamotrigine and polytherapy when Valproate was present in the regimen. There was no association with the dose. No specific malformation pattern recognized.

10. Levetiracetam:

It is not extensively metabolized, about 65% is excreted unchanged in urine.

Uses: In partial and myoclonic seizures and GTCS

Dose: Start with 500 mg twice daily, maximum dose – 3000mg / day.

Contraindications: Used cautiously in pre-existing psychosis / schizophrenia and in rhabdomyolysis.

Teratogenicity: According to UK and Ireland Epilepsy and Pregnancy Registry, 1st trimester exposure to Levetiracetam is associated with low risk of Major Congenital Malformation (0.7%). In the polytherapy group, the risk of Malformations were varied, with highest in those given with Valproate or Carbamazepine and lowest in those given with Lamotrigine.

11. Topiramate:

About 70% of the drug is excreted unchanged in urine. Its t ½ is 21 hours.

Uses: It is used in partial seizures, GTCS and in refractory epilepsy. It is also used in Bipolar Mood Disorder and in migraine prophylaxis.

Dose: Start with 50 mg/day in 2 divided doses and maximum dose is 400 mg/day.

Contraindications: In Sulfonamide hypersensitivity. Used cautiously in nephrolithiasis, hyperchloremic non–anion gap metabolic acidosis, angle closure glaucoma and hyperthermia.

Teratogenicity: A study by Daniel Mines et al showed that Topiramate use is associated with increased prevalence of oral clefts.

12. Lacosamide:

It is metabolized in liver by CYP3A4, CYP2C9 and CYP2C19. It's t1/2 is 13 hours. About 95% is excreted in urine.

Uses: In partial seizures

Dose: Start with 100 mg twice daily and maximum dose is 300 – 400 mg/day.

Hepatic and severe renal disease required 25% decreased in dose.

Contraindications: Used cautiously in Cardiac conduction abnormalities and in phenylketonuria.

Teratogenicity: Animal studies showed increased neurotoxicity.

13. Felbamate:

About 40% – 50% is excreted unchanged in urine. It's t1/2 is 13 – 23 hours.

Uses: In partial seizures and Lennox Gastaut syndrome

Dose: Start form 1200mg/day and maximum dose is 3600 mg/day.

Contraindications: In Carbamate hypersensitivity and aplastic anaemia.

Teratogenicity: No adequate studies available. But postmarketing surveillance reported microcephaly, GU malformations, anencephaly and fetal death.

14. Gabapentin:

It is highly lipid soluble and excreted unchanged in urine. Its t1/2 is 5 to 7 hours.

Uses: It is used as an adjuvant in partial seizures, painful neuropathies, Amyotrophic Lateral Sclerosis, Multiple Sclerosis, hot flushes in menopause and in Restless Leg Syndrome.

Dose: Start form 300 mg thrice daily to maximum dose of 3600 mg/day.

Contraindications: In renal impairment

Teratogenicity: No major malformations found with monotherapy.

15. Pregabalin:

It has a negligible hepatic metabolism

Uses: It is used as an adjuvant therapy in partial seizures, Generalized Anxiety Disorder and in Neuropathic pain.

Dose: Start with 150 mg/day and maximum dose is 600 mg/day.

Contraindications: In renal failure. Use cautiously in Congestive Heart Failure, Myoclonus and Thrombocytopenia.

Teratogenicity: Animal studies showed structural and neurodevelopmental problems and growth retardation.

16. Rufinamide:

Mainly metabolized in liver by hydrolysis and <2% is excreted unchanged in urine. Its t1/2 is 6-10 hours.

Uses: In refractory partial seizures. It is the only FDA approved drug for Lennox Gestaut Syndrome.

Dose: 3200 mg/day

Contraindications: In Familial short QT syndrome. It may cause diplopia, respiratory insufficiency and leukopenia.

Teratogenicity: Animal studies showed low birth weight, fetal visceral and skeletal problems.

17. Tiagabine:

It is metabolized in liver by CYP3A4 by oxidation and glucuronidation. Its t1/2 is 7–9 hours.

Uses: In partial seizures

Dose: Start with 4mg/day and maximum dose is 56 mg/day.

Contraindications: Used cautiously in hepatic disease.

Teratogenicity: Animal studies showed cranio facial, appendicular and visceral defects and low birth weight.

18. Vigabatrin:

It has no significant metabolism and mainly excreted through kidneys. Its t1/2 is 10.5 hours. Duration of action depends on rate of GABA synthesis rather than vigabatrine elimination.

Uses: Used in complex partial seizures and in infantile spasms.

Dose: 1.5 mg twice daily. Maximum dose is 3000mg/day.

Adverse effects: It can cause permanent B/L concentric visual field constriction.

Teratogenicity: Post marketing surveillance showed congenital cardiac defects, hydronephosis, vesicoureteric reflex, hip dysplasia, convulsions and renal dysplasia.

19. Zonisamide:

It is bound to RBC. Undergoes liver metabolism.

Uses: Adjuvant in partial seizures.

Dose: Start with 100 mg once daily and maximum dose is 600 mg/day.

Contraindications: Sulphonamide hypersensitivity

Adverse effects: Metabolic acidosis, hyperthermia.

Teratogenicity: Metabolic acidosis can have negative effect on fetus.

The following drugs need modification in renal disease:

- Levetiracetam, Topiramate, Felbamate, Gabapentin, Pregabalin and Vigabatrin

The following drugs require increase in the dose in pregnancy because of increased clearance:

- Lamotrigine, Levetiracetam, Carbamazepine, Oxcarbazepine and Phenytoin

The following drugs interfere with effectiveness of COC pills and implanted progestins because of their enzyme inducing property:

- Phenytoin, Phenobarbitone, Primidone, Carbamazepine, Oxcarbazepine, Eslicarbazepine, Clobazam, Rufenamide and Felbamate
- When the woman is using these enzyme inducing Anti-epileptic Drugs, though there is insufficient evidence about vitamin k supplementation in last month to the mother, routine vitamin k 1 mg intra muscular to neonate is recommended

The following drugs have no contraceptive failure when given along with combined oral contraceptive pills:

- Valproic acid, Ethosuximide, Lamotrigine, Levetiracetam, Lacosamide, Zonisamide, Vigabatrin, Gabapentin, Pregabalin and Tiagabine
- These drugs when used with Lamotrigine lower the blood levels of Lamotrigine which in turn increases the seizure frequency.

Common drug interactions:

Phenytoin concentration

- ↓ with carbamazepine and vice versa (increase in metabolism)
- ↑with valproic acid (displaces from protein binding site)

Anti-epileptic Drugs – Pharmacology & Teratogenecity

- ↑ with isoniazid (inhibits metabolism)
- also inhibits warfarin metabolism and vice versa

Valproate

- ↑ plasma concentration of phenobarbitone (inhibits metabolism) & phenytoin (displaces from protein binding)
- avoid valproate + clonazepam combination

Carbamazepine

- ↓ efficacy of oc pills, lamotrignine, topiramate (induces metabolism)
- ↓ efficacy of phenobarbitone, phenytoin, valproate & vice versa (induces metabolism)
- carbamazepine plasma concentration is ↑ by isoniazid (inhibits metabolism)

Lamotrigine levels

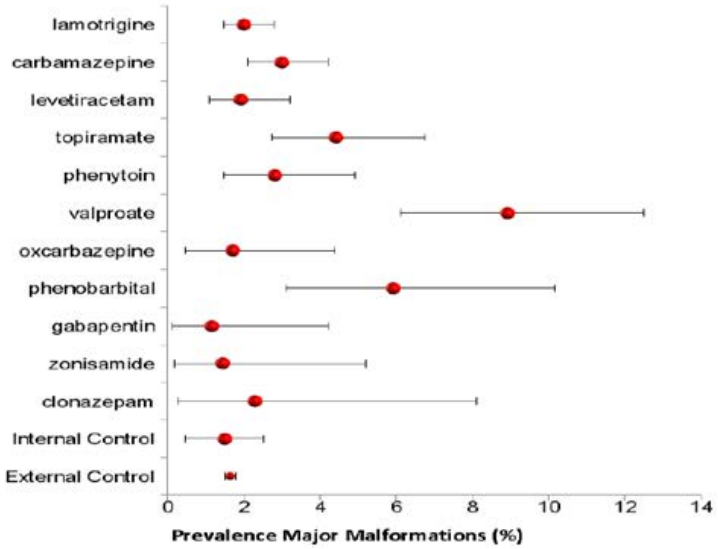
- ↓ by phenytoin, carbamazepine, phenobarbitone (↑ metabolism)
- ↑ by valproate (↑ metabolism)

Drugs for different types of seizures:

- **Focal seizures:** Carbamazepine, Oxcarbazepine, Lamotrigine, Levetiracetam and Valproate
- **Primary GTCS:** Valproate, Levetiracetam, Lamotrigine and Topiramate.
- **Absent seizures:** Valproate, Ethosuximide, Lamotrigine, Clonazepam and Clobazam
- **Myoclonic seizures:** Valproate, Lamotrigine, Levetiracetam, Topiramate, Zonisamide and Clonazepam
- **Atonic seizures:** Valproate, Clonazepam, Clobazam, Lamotrigine and Topiramate

Pregnancy Registries:

Prevalence of major malformations:



N	%	95% CI
2143	2.05%	(1.5 to 2.8)
1080	2.78%	(1.9 to 4.0)
999	2.00%	(1.3 to 3.1)
468	5.34%	(3.6 to 7.9)
427	2.58%	(1.4 to 4.7)
341	8.18%	(5.5 to 11.9)
266	2.63%	(1.2 to 5.6)
201	6.12%	(3.3 to 10.7)
201	1.49%	(0.4 to 4.7)
161	0.62%	(0.0 to 3.9)
100	2.00%	(0.3 to 7.7)
745	1.21%	(0.6 to 2.4)
69277	1.60%	(1.5 to 1.7)

Anti-epileptic Drugs – Pharmacology & Teratogenecity

The above table is from the North American Anti-epileptic Drug Pregnancy Registry.

The risk of Major Congenital Malformations are more with Valproate ((8.18%), followed by Phenobarbital ((6.12%) followed by Topiramate (5.34%). Malformations are least with Lamotrignine (2.05%) and Levetiracetam (2%).

EURAP Registry – International Registry of Anti–epileptic Drugs and Pregnancy:

According to this Registry, when compared 8 different anti – epileptic drugs, namely Valproate, Phenobarbital, Phenytoin, Carbamazepine, Oxcarbazepine, Topiramate, Levetiracetam and Lamotrigine,

The prevalence of Major Congenital Malformations are more with Valproate (10.3%), Phenobarbital (6.5%), Phenytoin (6.4%) and Carbamazepine (5.5%).

The risk of Malformations with Lamotrigine, Levetiracetam and Oxcarbazepine is similar to unexposed population.

Australian Pregnancy Registry:

It is the only Registry which has a comparative arm of Women With Epilepsy (WWE) not on Anti-epileptic Drugs in 1st trimester.

It was found that the risk of malformations in WWE not on AEDs in 1st trimester was 3.3%. The malformation rates with Lamotrigine, Levetiracetam and Topiramate as a part of monotherapy are 4.6%, 2.4% and 2.4% respectively and it was not statistically significant. The malformation rate for Topiramate as a part of polytherapy (14.1%) and for Valproate both for monotherapy (13.8%) and polytherapy (10.2%) was statistically significant.

UK Pregnancy Registry:

It showed that the risk of Major Malformations are more with Valproate (6.7%) followed by Topiramate (4.3%).

Pregnancy Registry in South India:

From Kerala, Sanjeev V et al, the risk of Major Congenital Malformations are more with polytherapy (9.9%) and monotherapy (6.4%) compared to Women With Epilepsy not on Anti–epileptic Drugs (5.6%) and general population (3.45%).

Among monotherapies, the risk is more with Valproate (8.96%) and Clobazam (22.2%).

In a recent systemic review and network meta-analysis of congenital malformations and perinatal outcomes in women on anti–epileptic drugs by Areti Angeliki Veroniki et al in 2017 showed:

- High risk of MCM compared to controls with Ethosuximide, Valproate, Topiramate, Phenobarbital, Phenytoin, Carbamazepine, Clobazam and Gabapentin.
- Lamotrigine and Levetiracetam are not associated with increased risk of malformations.
- No statistically significant increased risk to physical development with Levetiracetam, Lamotrigine, Oxcarbazepine and Vigabatrine.
- Significant association with fetal loss with Topiramate.
- Increased risk of growth restriction with Clobazam, Topiramate & Phenobarbital
- Increased risk of preterm birth with Clobazam.

NEAD STUDY (Neurodevelopmental Effects of Anti–epileptic Drugs)

The IQ for the child at the age of 6 years was lower if the mother used Valproate in the antenatal period compared to Lamotrigine, Carbamazepine and Phenytoin.

The children of women who breastfed their children while on anti-epileptic drugs had IQ 4 points higher than those who did not breastfeed.

Conclusion:

- Sodium valproate was more teratogenic compared to other antiepileptics.
- Congenital malformations are more with polytherapy than monotherapy.
- Most of the studies showed Lamotrigine, Levetiracetam and Oxcarbamazepine safer among all AEDs.
- Drug interactions should be kept in mind and appropriate dosage adjustments should be made.
- Breastfeeding is compatible with the commonly used AEDs.
- Never stop AEDs for fear of teratogenicity in 1st trimester, even valproic acid. Ideal is to change to another drug before conception.

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Approach to Headache in Pregnancy

– Dr. Bhagyasri G.
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Introduction:

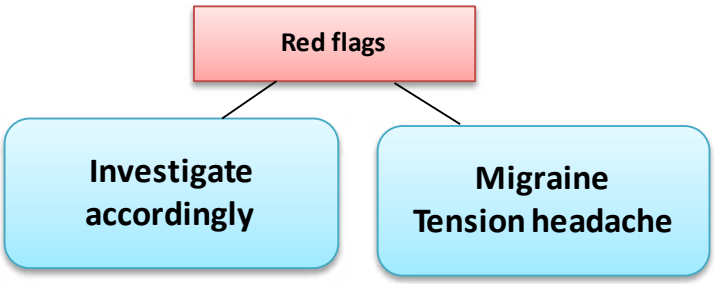
Headache is a common symptom among pregnant women. Differentiating between benign, often chronic, headache syndromes and acute conditions signifying more serious pathophysiology is crucial. A careful history and physical exam can often aid the clinician in distinguishing between chronic and acute headache syndromes in pregnant women. Likewise, diagnostic imaging and the appropriate use of medical therapy need to be tailored to pregnancy, to avoid known teratogenic, uterotonic or otherwise potentially harmful agents. Physiologic changes induced by pregnancy increase the risk of cerebral venous thrombosis, arterial dissection, and pituitary apoplexy. Preeclampsia, a serious condition unique to pregnancy, must also be considered.

Red flag features for potential secondary headache (adapted from Scottish Intercollegiate Guidelines Network guidance)

- **Thunderclap:** rapid time to peak headache intensity (seconds to 5 minutes), e.g. with a subarachnoid haemorrhage
- Focal neurological symptoms (e.g. limb weakness, aura <5 minutes or >1 hour)
- Non-focal neurological symptoms (e.g. cognitive disturbance) – seen in central venous thrombosis
- Change in headache frequency, characteristics or associated symptoms
- Abnormal neurological examination
- Headache that changes with posture – a sign of high or low cerebrospinal fluid pressure
- Headache awakening the patient – associated with migraine and raised intracranial pressure
- Headache precipitated by physical exertion or Valsalva manoeuvre – consider subarachnoid haemorrhage or raised intracranial pressure
- Patients with risk factors for cerebral venous thrombosis
- Jaw claudication or visual disturbance – associated with giant cell arteritis (women over 50 years)
- Fever – consider meningitis
- Neck stiffness – indicative of meningeal irritation
- New onset of headache in a patient with a history of HIV infection
- New-onset headache in a patient with a history of cancer

Approach to Headache in Pregnancy

Patient Evaluation	
History	**Physical examination**
Timeline	Fundoscopy
Location	Full neurological examination
Character	Plantar responses
Associated symptoms	Cranial nerve assessment
Relevant medical/ surgery/ psychiatric illness	Gait assessment
Medication history	BP, urine for protein and clonus



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Approach to Headache in Pregnancy

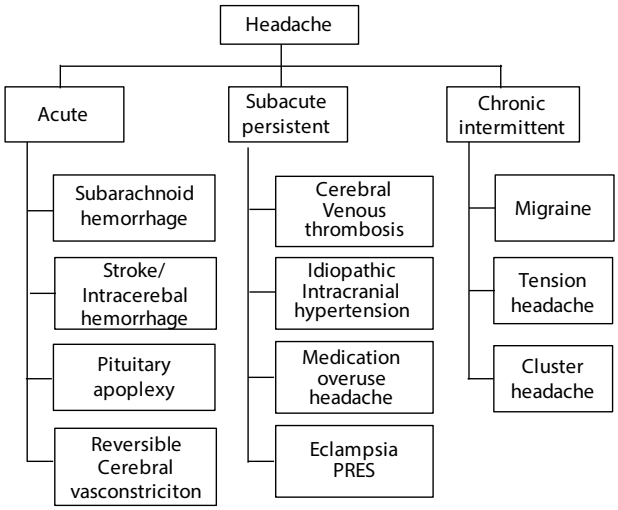

Fig. 1: Differential Diagnosis of headaches in pregnancy. PRES posterior reversible encephalopathy syndrome

Chronic intermittent headaches

Migraine: The condition is more common in women, with the highest prevalence rates during the childbearing years. However, pregnancy itself leads to a reduction in the frequency and severity of attacks of migraine without aura. A migraine is classically:

- Unilateral, Pulsating headache
- Aggravated by physical activity
- Associated with nausea and vomiting and/or
- Photophobia or phonophobia
- Preceding aura consisting of a reversible focal neurologic deficit

Cluster Headache:

It is usually severe and unilateral, orbital, supraorbital, and/or temporal, occurs mostly at night. It occurs up to eight times daily, in clusters, typically 6-12 weeks duration once a year/ 2 year and at the same time of the year. It is associated ipsilateral autonomic dysfunction (e.g., conjunctival injection, lacrimation, rhinorrhea, miosis, etc.). First-line treatment in pregnancy - high flow oxygen. In 60% - significant reduction in pain after 30 minutes of oxygen therapy. Sumatriptan, effective in 2/3 of CH patients or Ipsilateral intranasal lidocaine (pregnancy category B) has been used with rapid relief and is effective in 1/3 of CH cases. Dihydroergotamine is contraindicated in pregnancy

Rebound Headache:

It is due to sudden caffeine withdrawal daily > 200mg/ day. Caffeine withdrawal > 500mg intake (approx 300ml) may result in spontaneous abortions.

Sinus headache:

Due to increase in vascularity and mucus production, pregnancy is associated with an increased susceptibility to sinus congestion. Women who are prone to sinus congestion and sinusitis may note an increase in sinus headaches during pregnancy. They are typically periorbital or temporal and constant. Treatment of a sinus headache should include acetaminophen and decongestants, as well as antibiotic therapy if there are signs and symptoms of bacterial sinusitis, such as fever and tenderness to palpation over sinuses on physical examination.

Sub acute headaches

Headache attributed to Preeclampsia or eclampsia:

Headache has developed in temporal relation to the onset of the pre-eclampsia or eclampsia

Either or both of the following:

a) headache has significantly worsened in parallel with worsening of the pre-eclampsia or eclampsia

b) headache has significantly improved or resolved in parallel with improvement in or resolution of the pre-eclampsia or eclampsia

Headache has at least two of the following three characteristics:

a) bilateral location

b) pulsating quality

c) aggravated by physical activity

Postdural puncture headache:

Puncture of the dura occurs in 0.5–2.5% of epidurals. If accidental dural puncture occurs with an epidural needle there is a 70–80% chance of a postdural puncture headache. The headache is usually in the fronto-occipital regions and radiates to the neck. It is characteristically worse on standing and typically develops 24–48 hours post-puncture. Conservative management includes hydration and simple analgesics Untreated, the headache typically lasts for 7–10 days but can last upto 6 weeks. Epidural blood patch has a 60–90% cure rate.

Approach to Headache in Pregnancy

Posterior reversible encephalopathy syndrome(PRES):

Posterior reversible encephalopathy syndrome (PRES) is a clinical–neuroradiological entity associated with pre-eclampsia. It is characterised by headache, vomiting, visual disturbances, seizures and altered mental state, with radiological findings of oedema in the posterior circulation of the brain breakdown of the normal blood–brain barrier and culminating in vasogenic brain oedema. Because of a partial lack of sympathetic innervations of the vasculature that emerges from the basilar artery, oedema tends to occur in the posterior regions of the central nervous system. Oedema leads to progressive brain compression within the skull and the symptoms of headache, nausea, vomiting and seizures. eported associated with immunosuppressant drug use, nephrotic states, sepsis and systemic lupus erythematosus. In these cases a common aetiological pathway leading to vasogenic oedema is endothelial damage. Management includes blood pressure control, prevention and/or treatment of seizures and prompt delivery of the baby.

Cerebral venous thrombosis:

Pregnancy is a recognised risk factor for the uncommon but catastrophic event of cerebral venous thrombosis (CVT), perhaps as a result of prothrombotic changes and dehydration. Caesarean section, systemic infection, vomiting and anaemia increase the risk. The greatest risk period is the third trimester and the first 4 weeks postpartum. Thrombosis of the sagittal sinus with secondary extension into the cortical veins, or primary thrombosis of one of the cortical veins, are the most common sites of involvement in pregnancy. Headache is the most frequently (80–90%) occurring symptom in cerebral venous thrombosis and often the first symptom reported by patients. Headache with focal neurologic findings or seizures should increase suspicion of CVT. MRI/MRV brain is investigation of choice. Anticoagulation is the standard of care.

Idiopathic Intracranial Hypertension:

Idiopathic intracranial hypertension is a rare condition but more prevalent in obese women of childbearing age. It may present for the first time in pregnancy and pre-existing disease tends to worsen during pregnancy. The headache is generalised, non-throbbing, aggravated by coughing or straining and is associated with diplopia (38%) and visual loss (31%) with papilloedema. Diagnosis requires excluding other causes and finding abnormally elevated cerebrospinal fluid pressure (>20 cm H2O) on lumbar puncture. Management includes monitoring of the visual fields andvisual acuity because of the risk of optic nerve infarction. Women should be encouraged to limit weight gain. Treatment with therapeutic lumbar puncture or acetazolamide (500 mg twice daily) is directed towards improving the headache and preventing visual loss. Incidence 5% in the pregnant population, 0.9 per 100,000 in the general population, 4-19 per 100,000 in obese women. Visual outcomes same as those of nonpregnant women. pregnancy outcome is unaffected.

Management includes diet and weight control, serial LPs and/or acetazolamide (category C). Some data suggest acetazolamide can be safely used in pregnancy; dosing starts at 0.5-1g/ day in divided doses to a maximum of 2g/day. Steroids (category C) are reserved for urgent short-term treatment in patients awaiting surgery.

Surgical therapy - if severe or progressive visual loss despite medical Management.

- Optic nerve sheath fenestration
- Lumboperitoneal or ventriculoperitoneal shunting

Over 50% of shunt become occluded, infected, or migrate requiring reoperation.

Acute headaches

Subarachnoid haemorrhage:

Subarachnoid hemorrhage occurs most commonly as the result of a ruptured aneurysm or arterial-venous malformation (AVM). In pregnancy, AVM rupture typically occurs early (15- 20 weeks GA) and in younger women (20-25 years), while aneurysm rupture usually occurs later (30-40 weeks GA). and in older women (30-35 years). Patients will classically present with a thunderclap headache, but nausea and vomiting, stiff neck, photophobia, syncope, and focal neurologic deficit may also be seen. Patients with a preceding less severe headache episode should raise concern for a sentinel bleed.

Evaluation of SAH should begin with a non-contrast head CT. If negative, a LP should be performed and the CSF examined for xanthrochromia. If necessary, CT angiogram (CTA) may localize the lesion (e.g. aneurysm, AVM) or document vasospasm. Patients with SAH should be hospitalized to facilitate early neurosurgical intervention with clipping or coiling to reduce the risk of rebleeding.

Pituitary Apoplexy:

Pituitary apoplexy is caused by acute ischemic or hemorrhagic infarction as the pituitary gland expands and outgrows its blood supply or compresses the vessels against the sella. It is most commonly seen in men over the age of 50 years and in a pre-existing pituitary adenoma. Although rare, the massive hyperplasia of lactotrophs that occurs in pregnancy causes the pituitary gland to grow by as much as 130% putting pregnant patients at risk. Patients will present most commonly with sudden onset severe headache and nausea and vomiting and less commonly with visual symptoms, altered mental status, or coma. Secondary adrenal insufficiency can occur, causing severe hypotension and hyponatremia which can be life-threatening. In pituitary apoplexy, CT may demonstrate a recent bleed or hyperdense lesion in the pituitary. MRI is more sensitive for delineating the relationship between the pituitary and surrounding structures. Pituitary apoplexy patients with persistent visual symptoms, neurologic deficit, or altered mental status require urgent surgical decompression. Secondary adrenal insufficiency should be treated immediately with fluid and electrolyte replacement and hydrocortisone.

Reversible cerebral vasoconstriction syndrome (RCVS):

Reversible cerebral vasoconstriction syndrome (RCVS) is a cerebrovascular disorder associated with multifocal arterial constriction and dilation. It has a significant association with the postpartum period. RCVS is characterised by recurrent sudden onset and severe headaches over 1–3 weeks, often accompanied by nausea, vomiting, photophobia, confusion and blurred vision. Diagnosis requires the demonstration of diffuse arterial beading on cerebral angiography with resolution within 1–3 months. It is in the differential of a postpartum thunderclap headache often made after subarachnoid haemorrhage has been excluded but the headaches recur. Treatment is currently based on expert opinion including the use of calcium channel blockers, high-dose corticosteroids and magnesium sulphate.

Meningitis:

Meningitis in pregnancy presents similarly to that in the non-pregnant population with headache, fever, nausea, vomiting, nuchal rigidity, and/or altered mental status. Otitis and sinusitis infection often precede meningitis in pregnancy. Streptococcus pneumoniae and Listeria monoctogenes are the most common causative organisms and are associated with a very high mortality rate (28%). Miscarriages and neonatal death are also common consequences of meningitis in pregnancy.

If meningitis is suspected, blood cultures and a LP should be obtained. A CT of the head should be done prior to the LP if there are any concerns for elevated ICP. Studies suggest that once antimicrobial therapy is started, sterilization of the CSF occurs within two hours for N. meningitides infections, and within four hours for S. pneumoniae infections; however, antibiotics should not be delayed if LP cannot be performed early.

Empiric antimicrobial therapy for meningitis consists of a third-generation cephalosporin, such as cefotaxime or ceftriaxone (category B), and vancomycin (category C). Because Listeria infection is common in pregnancy, ampicillin (category B) should also be given. If viral causes are suspected, add acyclovir (category B). Studies suggest that prognosis in viral meningitis is directly related to the delay in empiric acyclovir administration. Some data suggest adjunct therapy with steroids reduces mortality. Dexamethasone has few side effects in third trimester pregnancy (category C) and may also be used.

Summary:

- History, physical examination and appropriate investigation as needed according to the algorithm
- Acute headache with red flag signs consider for brain imaging, specialist consult and emergency treatment with mother as the priority

References:

1. Kirsty Revell, Paul Morrish. Headache in pregnancy. RCOG TOG 2014
2. Schoen J, Campbell R, Sadosty A. Headache in Pregnancy: An Approach to Emergency Department Evaluation and Management.Western Journal of Emergency Medicine; Volume XVI, NO. 2 : March 2015.

NEWSLETTER Obstetric Medicine

Epilepsy – Etiology and Classification

– Dr. Vinita Udupa
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Epilepsy is the most common neurological complication of pregnancy with a prevalence of 0.5-1%. Women with epilepsy are at substantially increased risk of adverse outcomes like hypertensive disorders, preterm labour, stillbirth, CS and a 10-fold increased risk of maternal death. Majority of these deaths which are classified as SUDEP (sudden unexpected death in epilepsy) occur in those women with a poor seizure control. Anti-epileptic medications also pose a separate risk of congenital malformations in the fetus. Thus, balancing the need of these medications versus their risk of teratogenicity is an important aspect of management of women with epilepsy.

Definition:

A seizure is defined as a transient occurrence of signs and/or symptoms due to an abnormal excessive or synchronous neuronal activity in the brain. Whereas, epilepsy represents a diverse array of brain diseases sharing the common presentation of seizures.

However, the practical use of these definitions was difficult and thus the International League Against Epilepsy (ILAE) came up with 3 separate operational definitions of epilepsy in 2017 that are more clinically applicable.

1. At least 2 unprovoked seizures occurring greater than 24 hours apart
2. One unprovoked seizure and the probability of further seizures similar to the general recurrence risk (>60%) after 2 unprovoked seizures, occurring over next 10 years
3. Diagnosis of an epilepsy syndrome

Classification:

Classifying epilepsy is important in several ways. It provides for a common terminology for communication among all clinicians and researchers and allows for grouping of patients for therapy. Also, it enables researchers to focus on the mechanism of seizure types.

In 1989, the ICE (International classification of epilepsy and epilepsy syndromes) had introduced a classification of seizures of 2 divisions. Generalised versus partial/ focal and secondly epilepsy with known etiology versus idiopathic and cryptogenic.

This classification has been highly influential worldwide and has had a major impact on clinical practice and research. It has been valid for the last 3 decades and is still in vogue. However, a revision was needed to account for subsequent scientific discoveries as the old classification did not include many types of seizures.

Thus, in 2017, ILAE developed a new revised classification.

Important changes included – Extinction of the terms partial and complex, instead only describing the presence of awareness, addition of motor and non-motor classification of focal seizures and addition of a combined focal and generalised seizure category and an unknown seizure category type.

This classification is a multi-level classification which allowed for diagnosis at 3 levels according to the range of resources that may be available.

1. Seizure type
2. Epilepsy type
3. Epilepsy syndrome

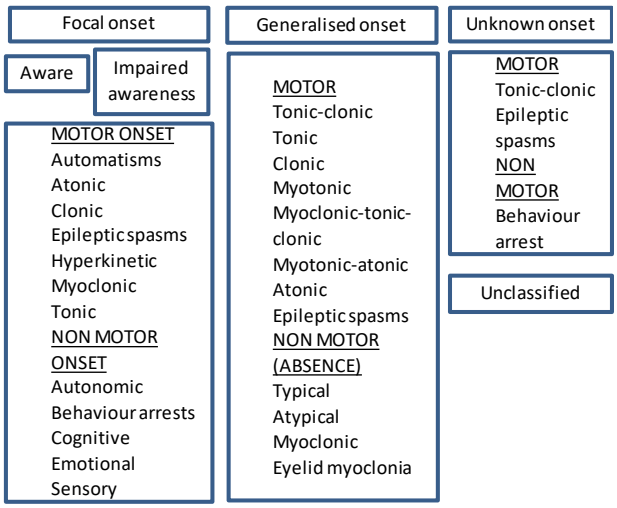
In low resource areas, diagnosis may be limited to level 1, whereas in tertiary centres, a seizure may be considered in among all levels of diagnosis.

1. Seizure type:

First step is to separate seizures by how they begin in the brain i.e. whether the initial manifestation of the seizure is focal or generalised

- Focal onset
- Generalised onset
- Unknown onset

The expanded classification of various types of seizures is tabulated below.



Epilepsy – Etiology and Classification

In the new classification, awareness has been used instead of consciousness as it is simpler to evaluate and is practically important as patient's safety is involved. Focal seizures may or may not present with intact awareness, whereas generalised seizures are always considered to have impaired awareness.

Generalised seizures are those that begin simultaneously in both sides of the brain. They can be either motor or non motor. Generalised non-motor are primarily absence seizures.

2. Epilepsy type:

The second level of classification is based on the type of epilepsy, as follows:

- Generalised epilepsy
- Focal epilepsy
- Combined generalised and focal epilepsy
- Unknown category

3. Epilepsy syndrome:

The final level of classification should ideally be able to evaluate a possible underlying syndromic diagnosis of the seizures. The pattern of features enabling the diagnosis are seizure type, EEG findings and imaging abnormalities.

Etiology of Seizures:

Determining the etiology should be the main aim from the first epileptic seizure. ILAE in 2017 divided various seizures into 6 different etiological types.

1. Structural
2. Genetic
3. Infectious
4. Metabolic
5. Immune
6. Unknown

The 1st investigation should be neuroimaging, ideally MRI, to differentiate structural etiology from the rest. A patient's epilepsy may be classified into more than one etiological category e.g. Tuberous sclerosis which has both a structural and genetic etiology.

1. Structural causes:

In this type, a structural abnormality has a substantially increased risk of being associated with seizures. These abnormalities are mostly visible on neuroimaging. Examples include genetic conditions like malformations of the cortical development or acquired ones like stroke, trauma, HIE, infection. The advantage of these type of seizures is that if medical therapy fails, epileptic surgery can be considered.

2. Genetic etiology:

This type of epilepsy directly results from a known genetic mutation in which seizures are a core symptom of the disorder. Epilepsies with these etiology are quite diverse. Many of the underlying genes are not known yet.

Genetic etiology can also have an environmental contribution like seizures exaggerated by sleep deprivation, stress, illness.

3. Infectious etiology:

This is the commonest etiology worldwide where known infections can manifest as seizures. Examples include: neurocysticercosis, TB, HIV, cerebral malaria, subacute sclerosing panencephalitis, cerebral toxoplasmosis, Zika virus, CMV infection. Similar to structural cause, these seizures also have specific treatment implications.

4. Metabolic etiology:

A range of metabolic disorders are associated with epilepsy. These are well delineated metabolic defects with manifestations or biochemical changes throughout the body. Porphyria, uremia, amino-acidopathies, pyridoxine dependent seizures are few examples. Many of these disorders also have a genetic defect. Identification of specific metabolic causes of epilepsy is extremely important for the management.

5. Immune etiology:

These type of seizures are due to autoimmune-mediated CNS inflammation. The main examples are anti-NMDA receptor encephalitis, anti-LGI1 encephalitis. These seizures can be treated with targeted immunotherapies.

6. Unknown causes:

For several type of seizures, it is difficult to make a specific diagnosis apart from the basic electroclinical semiology like Frontal lobe epilepsy. Extent of diagnosis in these cases depends on extent of resources available for evaluation.

NEWSLETTER Obstetric Medicine

Management of Stroke in Pregnancy Old and New

– Dr. Swathi M.
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Initial stroke management of pregnant women does not differ from that of the non-pregnant patient, with care focused on adequate oxygenation,maintaining circulatory integrity and euglycaemia.

Further management is then directed to con?rm whether stroke is ischemic or hemorrhagic as this will determine whether thrombolysis/clot retrieval is appropriate

1) Ischemic stroke:

Thrombolytic therapy:

- Stroke thrombolysis improves outcome for ischemic stroke for individuals presenting up to 4.5 h after onset of symptoms.
- Historically pregnancy was considered a relative exclusion for thrombolysis in ischemic stroke
- Thrombolysis
- The risks include-Preterm labour, Placental abruption, Fetal death, teratogenicity, PPH
- Can be considered in pregnancy,in moderate or severe stroke,where benefits outweighs the risks
- Recombinant tissue plasminogen activator (rt-PA) Alteplase(0.9mg/kg) is the current medication for ischemic stroke thrombolysis.
- The drug rt-PA is not known to be teratogenic, and the molecule is too large (72 000 kD) to cross the placenta.

Thrombectomy:

- Mechanical thrombectomy-
- Patients that present within 6 h of new onset of stroke symptoms , who present with an arterial occlusion in the proximal anterior circulation

Mechanical thrombectomy is an option for patients in whom Thrombolysis is contra-indicated

Statins:

- Statins have a well-established use for secondary stroke prevention

Currently, statins are considered to be contraindicated in pregnancy due to higher rates of birth defects.

2) Haemorrhagic stroke:

- Initial management of hemorrhagic stroke involves -medical (BP, seizures,cerebral edema, temperatue) if necessary, surgical intervention.
- BP management - requires careful assessment as increase in blood pressure may cause hemorrhage expansion.
- Conversely an increased MAP is required to maintain cerebral perfusion in some
- so a fall in systolic blood pressure may cause ischemia and worsen neurologic injury (subarachnoid hemorrhage, intracerebral hemorrhage)
- The overall goal is to minimize the risk of re bleeding.
- Pregnancy should not be regarded as a contraindication for angiography and endovascular treatment of a vascular cause for hemorrhage.
- For intracerebral hemorrhage, priority should focus on managing blood pressure, and on identifying and correcting coagulopathies.

Aneurysms:

- Unruptured cerebral aneurysm-Treatment deferred to post partum period in stable patients.
- Ruptured aneurysms- Interdisciplinary approach including neurosurgeon endovascular interventionalist, neurologist, Obstetricians

Coils or clip the ruptured aneurysms

AVM:

a. Management of unruptured AVM (without bleeding) requires an individualized approach.

b. Low grade symptomatic AVM amenable to surgical resection should be resected

c. The treatment timing of high grade AVM, requiring multimodality approach with an interdisciplinary team, including neurosurgery, neurology and expertise in maternal fetal medicine.

Mode of Delivery:

- Stroke is not an absolute contraindication to vaginal birth.
- Shared decision with the obstetrics team, neurology team, and consideration of patient preference is required
- Cesarean delivery may be necessary for standard obstetric indications (e.g., maternal history, fetal presentation, fetal status).
- For women who sustain a stroke in pregnancy, long term postpartum follow up should be made

including management for secondary prevention of stroke recurrence.

- Pre-pregnancy counseling should be offered to all women prior to a future pregnancy screening pregnant patients who experience a stroke for signs and symptoms of depression, especially those patients with previous history of mood disorders

Postnatal Thromboprophylaxis:

- compression stockings-ineffective in reducing VTE in stroke patients.
- LMWH-after haemorrhagic stroke excluded
- who have-1)Restriction of mobility

2) H/O VTE

3) Dehydration or comorbidities

Contraception:

- Combined Oral contraceptive pills to be avoided
- Progestin only pills,Mirena,Depo Provera,Implanon-safe

Management of pregnancy with previous History of stroke:

- Cause of initial stroke
- Desire for pregnancy
- Residual neurological deficits
- Preconception counselling
- stroke Prophylaxis
- Increased fetal surveillance in pregnancy may be required in women with a history of stroke given the likelihood of underlying vascular disease increases FGR risk
- Antenatal anesthesia consultation, and the creation of an anesthetic plan of care, is warranted to avoid any uncertainty regarding neuraxial anesthesia in labour

Mode of delivery:

- In women who have had a prior ischemic stroke vaginal delivery can be encouraged providing there is no obstetric contraindication.
- Women who have received prior treatment for an AVM or an aneurysm can also be reassured that vaginal delivery is appropriate.

Psychosocial care and counselling:

- Stroke can have severe effects on Mothers physical, psychological, cognitive health, in terms of quality of life after an ischemic stroke
- Multi-disciplinary neuro-rehabilitation and referral to specialist perinatal mental health services is essential for her capacity to care for her newborn child.
- Social interventions such as daily support and home adaptations may be appropriate

References:

1) Consensus statement by the Canadian Stroke Best Practices Stroke in Pregnancy Writing Group 2018

2) European Stroke Journal 2018, Vol. 3(3) 227–236 ! European Stroke Organisation 2018

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NEWSLETTER

Obstetric Medicine

SOCIETY OF
OBSTETRIC MEDICINE
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Aims and Objectives

- To establish a forum for exchange and interchange of views and for enhancing fellowship among its members and to come out with evidence-based guidelines for management of medical disorders complicating pregnancy.
- To assist in the establishment of Obstetric Medicine as an important sub-specialty to advance clinical and scientific knowledge of medical illness complicating pregnancy.
- To promote research and training in Obstetric Medicine.
- To foster collaboration with other regional and international societies interested in Obstetric Medicine.
- To carry out all such activities as would contribute to the promotion of Obstetric Medicine.
- To promote formation of patient forums.

Inviting Articles for Future Issues

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