


Basics of Mental Health and Clinical Psychiatry
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Lecture 38
Mood Stabilizer

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Hello everyone, let us start lecture number 38 that is Mood Stabilizers. So in the previous mood stabilizer that we will be discussing are lithium, valproate, lamotrigine, carbamazepine and topiramate.

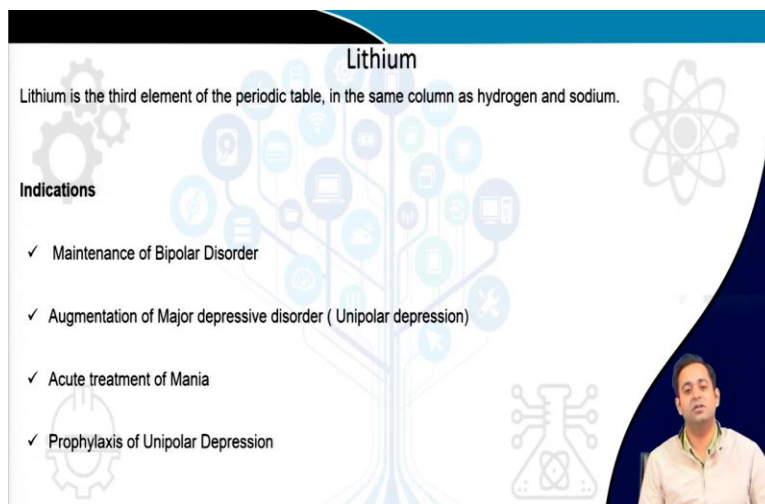
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Mood Stabilisers

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The slide features a central tree diagram where the trunk and branches are composed of various icons related to technology, medicine, and science, such as a smartphone, a laptop, a brain, a gear, and a chemical flask. The background is white with a blue header and footer. A presenter is visible in a small window on the right side of the slide.




Lithium

Lithium is the third element of the periodic table, in the same column as hydrogen and sodium.

Indications

- ✓ Maintenance of Bipolar Disorder
- ✓ Augmentation of Major depressive disorder (Unipolar depression)
- ✓ Acute treatment of Mania
- ✓ Prophylaxis of Unipolar Depression

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This slide is similar to the first one, with a tree diagram of icons. It includes a definition of Lithium and a list of its clinical indications. The presenter is also visible in the bottom right corner.

Other uses of Lithium (Not FDA approved)

- ✓ Lithium is also used to treat aggressive and self-mutilating behaviour, and recent studies
- ✓ raise the white blood cell (WBC) count in patients receiving clozapine.
- ✓ Used to prevent & treat steroid induced psychosis
- ✓ Antisuicidal Properties

Neuroprotective

1. Lithium may have neuroprotective effects that preserve the function of neurons and neuronal circuits.
2. Lithium also promotes the creation of new neurons (neurogenesis) in the hippocampus, which is potentially important for learning, memory and stress responses.

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So mood stabilizers are basically the ones which are most commonly indicated for the treatment of bipolar disorders in psychiatry, bipolar disorders, major depressive episodes of depression, or certain behavioral issues, agitations, and yes, most primarily indicated for seizures that is a epilepsy.

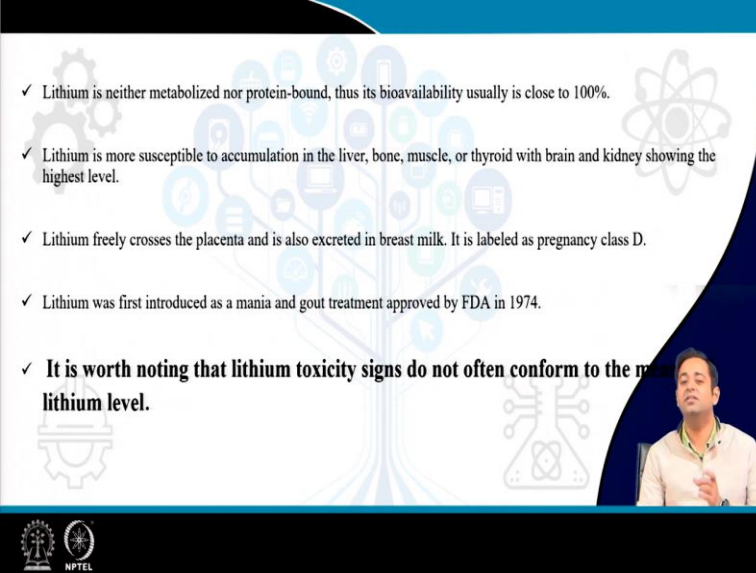
So let us start with lithium first, lithium is actually the classic mood stabilizer which is used in psychiatric illnesses. This is a third element of the periodic table which is placed aside hydrogen. What are the indications? Most commonly it is indicated for bipolar disorder, maintenance of bipolar disorder, augmentation of major depressive illnesses that is unipolar depression, acute treatment of mania, and prophylaxis of unipolar depression.

This particular model molecule was discovered by John F Kate of Australia. So this was actually encountered in the hot water springs where various other soils of lithium were found that is lithium carbonate, lithium citrate, lithium orotate, so various other soils are present. What are the other uses of lithium, which are not FDA approved? These are to treat that is aggressive episodes or nucleative behaviors.

It raises the white blood cell count on patients who are giving, who are on close up in. They are also used to prevent steroid induced psychosis and has a very important anti-suicidal property. Among neuroprotective role of lithium it is, because it preserves the various neurons in your

dendritic processes and in the neural circuits, it promotes the creation of neurogenesis in the hippocampus, which is potentially important for learning memory and stress responses.

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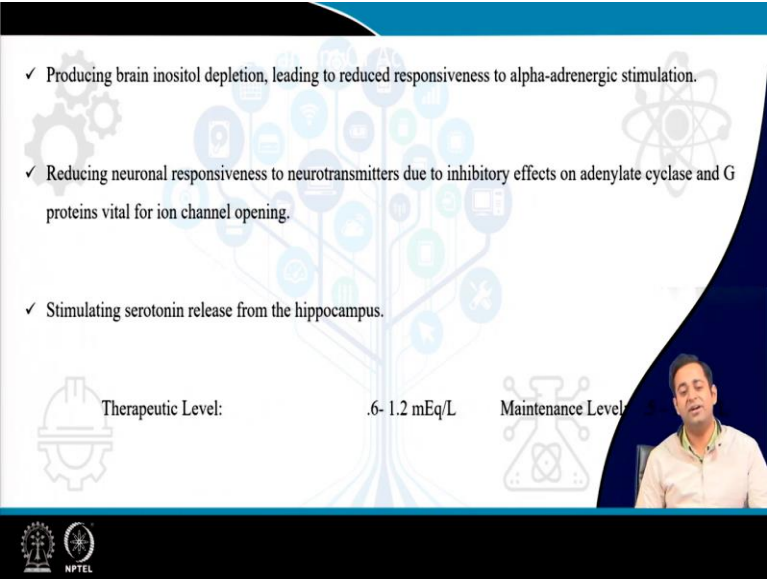


- ✓ Lithium is neither metabolized nor protein-bound, thus its bioavailability usually is close to 100%.
- ✓ Lithium is more susceptible to accumulation in the liver, bone, muscle, or thyroid with brain and kidney showing the highest level.
- ✓ Lithium freely crosses the placenta and is also excreted in breast milk. It is labeled as pregnancy class D.
- ✓ Lithium was first introduced as a mania and gout treatment approved by FDA in 1974.
- ✓ **It is worth noting that lithium toxicity signs do not often conform to the normal lithium level.**

So it is neither metabolized or not protein bound, thus if having a bioavailability of 100 percent. It is more susceptible to accumulating liver, bone, muscle, thyroid, brain, kidney that is why we need to have repetitive investigations pertaining to thyroid levels or kidney function test or liver function tests.

So lithium, it crosses placenta very easily and is excreted in breast milk also and that is why is labeled as pregnancy class D. And the most peculiar feature of lithium is that, the signs and symptoms of lithium toxicity are visible even though the serum lithium levels of, serum lithium levels in the blood is within the control or in the maintenance level.

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✓ Producing brain inositol depletion, leading to reduced responsiveness to alpha-adrenergic stimulation.

✓ Reducing neuronal responsiveness to neurotransmitters due to inhibitory effects on adenylate cyclase and G proteins vital for ion channel opening.

✓ Stimulating serotonin release from the hippocampus.

Therapeutic Level: .6- 1.2 mEq/L Maintenance Level

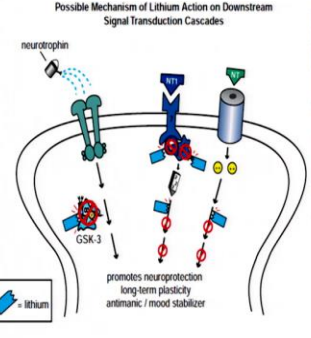
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So that needs to be the ones the clinicians are should be very much should be very much aware of this particular fact and that the side effects are like it is most obviously expressed out even before the serum levels are beyond the range, therapeutic range. So lithium reduces neuronal responsiveness to neurotransmitters due to inhibitory effects of adenine cyclists, G proteins, vital for iron channel opening.

And it stimulates serotonin release from the hippocampus. It produces brain anastrozole depletion leading to reduce responsiveness to alpha adrenergic stimulation. The therapeutic level is 0.6 to 1.2 and the maintenance level is 0.5 to 1 milliequivalent per liter. The dosage for which it should be having 0.6 to 1.2 is 600, 600 to 800 milligrams and for 900 to 1200 milligrams it should be 0.8 to 1.2 milliequivalent per liter.

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Mechanism Of Action: Lithium



Possible Mechanism of Lithium Action on Downstream Signal Transduction Cascades

1. Signal transduction sites which includes second messengers, phosphatidylinositol system, where lithium inhibits the enzyme inositol monophosphatase
2. Modulation of G proteins
3. Regulation of gene expression for growth factors and neuronal plasticity by interaction with downstream signal transduction cascades including inhibition of GSK-3 (glycogen synthase kinase-3) and protein kinase C.

promotes neuroprotection
long-term plasticity
antimanic / mood stabilizer

Legend:  = lithium

What are the mechanism of action? First is signal transduction various cascading effects, second is by modulation on the G proteins, and third is by the neutral neurotopins, those growth factors which mediate neural plasticity.

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Drug interactions with lithium

- ✓ Angiotensin-converting enzyme (ACE) inhibitors can
 - a) Reduce thirst leading to mild dehydration
 - b) Increase renal sodium loss leading to increased Na re-absorption by the kidney, resulting in an increase in lithium plasma levels.
- ✓ Diuretics can reduce the renal clearance of lithium, the magnitude of this effect being greater for thiazide diuretics. Lithium levels usually rise within 10 days of a **thiazide diuretic** being prescribed.

So how does it interact with other molecules. So AC inhibitors are the ones, it has a very important role. It reduces thirst leading to mild dehydration. It will increase the renal sodium loss leading to increased sodium reabsorption by the kidney, resulting in an increase in lithium

plasma levels. Diuretics can reduce the renal clearance of lithium, that is thiazide diuretics have a prominent role in this.

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Pre treatment work up for Lithium

- ✓ Pre-lithium workup eGFR and TFTs. ECG recommended in patients who have risk factors for, or existing cardiovascular disease.
- ✓ Baseline measure of weight desirable.
- ✓ Blood should be taken 12 hours after the last dose.
- ✓ Monitoring Plasma lithium every 6 months

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There has to be a pre-treatment workup of lithium before the lithium is started for a patients that is globular filtration rates the thyroid function tests and ECGs are recommended primarily before starting the lithium molecule and there should be a, the test should be repeated within six months.

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Signs and Symptoms Of Lithium toxicity

□ Mild & Moderate Intoxication (1.5-2.0mEq/L)

- Gastrointestinal – Vomiting
 - Abdominal Pain
 - Dryness of Mouth
- Neurologic-
 - Ataxia
 - Dizziness
 - Slurred of Speech
 - Nystagmus
 - Muscle Weakness

The slide features a background graphic of a tree with various medical icons as leaves. A video inset in the bottom right corner shows a male speaker. The NPTEL logo is in the bottom left corner.

So there are three stages for lithium toxicity which should be assessed very properly. So that the patient can be saved from lithium toxicity, these are mild, moderate, and severe toxic. In case of mild, toxicity of lithium that is gastrointestinal and logical manifestations among gastrointestinal manifestations the patient might be suffering from vomiting, abdominal pain, dry mouth. And neurological manifestations are ataxia, and dizziness, slurred of speech, nystagmus, muscle wasting.

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Signs and Symptoms Of Lithium toxicity

- ☐ **Moderate & Severe Intoxication (2.0-2.5 mEq/L)**
 - Gastrointestinal - Anorexia
 - Persistent nausea & Vomiting
 - Neurologic
 - Blurred Vision
 - Fasciculations
 - Hyperactive Deep Tendon reflexes
 - Convulsions
 - Delirium
 - Stupor
 - Coma
 - Circulatory Failure (lowered BP , Cardiac Arrythmia)
- ☐ **Severe Lithium Intoxication (Lithium Level >2.5 mEq/L)**
 - Generalised Convulsions
 - Oliguria and Renal Failure
 - Death

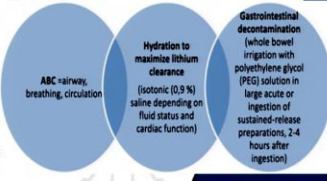
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In case of moderate or severe intoxication, you have anorexic. Patient can become anorexia, persistent nausea, or vomiting, can be blurred vision, convulsions, deep tendon reflexes are hyperactive, super delirium. And in case of severe serum lithium levels, where it is more than 2.5 the patient can have convulsions, oliguria and renal failure leading to death.

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Treatment

- Lithium has a very narrow therapeutic index, and toxic levels are when the drug is above 2 mEq/L.
- Lithium toxicity has no antidote.
- Treatment for lithium toxicity is **primarily hydration** and to stop the drug.
- Give hydration with normal saline, which will also enhance lithium excretion.
- Avoid all diuretics. **Gastrointestinal Decontamination**
- If the patient has severe renal dysfunction or failure, or severely altered mental status **hemodialysis**.
- 20 to 30 mg of propranolol given 2 to 3 times per day may help reduce tremors.



ABC = airway, breathing, circulation

Hydration to maximize lithium clearance (isotonic (0.9 %) saline depending on fluid status and cardiac function)

Gastrointestinal decontamination (whole bowel irrigation with polyethylene glycol (PEG) solution in large acute or ingestion of sustained-release preparations, 2-4 hours after ingestion)

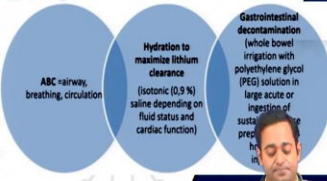
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So how can we treat this problem since lithium does not have any antidote primarily we should go for supportive therapy and the primary hydration of the patient. So avoid all, diuretics gastrointestinal decontamination should be done and if the left serum levels of lithium has gone beyond four mille equivalence per liter then the hemodialysis should be done for the patient.

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Valproate

- ✓ Most widely prescribed mood stabiliser in psychiatry.
- ✓ Its called valproic acid because it is converted into acidic form in the stomach.
- ✓ Metabolized by hepatic glucuronidation & mitochondrial beta oxidation.

**Plasma half – life- 10 -16 hrs:
Therapeutic dose range- 20-30 mg/kg body weight**

Therapeutic Indications

- Acute Mania
- Acute bipolar depression
- Prophylaxis of depression both(unipolar & bipolar)
- Augmentation of antipsychotics in treatment of schizophrenia & Schizoaffective disorder
- Behavioural agitation , Personality disorder , dementia with Behavioural & psychological sympt



It is come to sodium valproate, it is most commonly mood stabilizer which is used in psychiatry and it is metabolized by hepatic, glucuronidation and mitochondrial beta oxidation. Plasma half-life is from 10 to 16 hours and the therapeutic dose ring for the molecule is desired by 20 to 30 milligram per kg body weight.

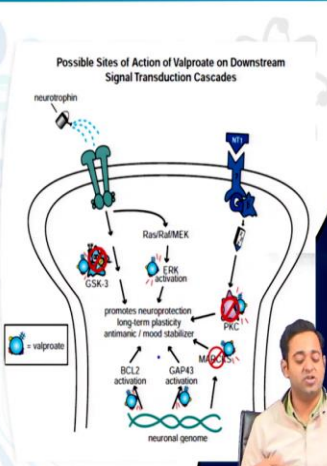

The indication is with acute mania bipolar depression, prophylaxis of the bipolar disorder, augmentation of any antipsychotic given for a psychiatric illness, a behavioral agitation personality disorders dementia with behavioural and psychological symptoms.

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Mechanism Of Action: Valproate

- ✓ Enhancement of GABA activity.
- ✓ Modulation of voltage sensitive sodium channels multiple downstream effects on signal transduction cascades, which may be involved in its antimanic effects.
- ✓ Valproate inhibits glycogen synthase kinase 3 (GSK-3), protein kinase C (PKC) and myristolated alanine-rich C kinase substrate (MARCKS).

Possible Sites of Action of Valproate on Downstream Signal Transduction Cascades

Mechanism of action of valproate is by enhancement of GABA activity. Second is by modulation of voltage sensitive sodium channels by various downstream effects on signal transduction cascades. And thirdly is by those glycogen synthase inhibition of glycogen synthase, 3 inhibitors and protein kinase 3 inhibitors.

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Common adverse effects

Nausea , vomiting , dyspepsia , diarrhoea , sedation , ataxia , tremors ,dysarthria , weight gain , hair loss.

Neuroprotective

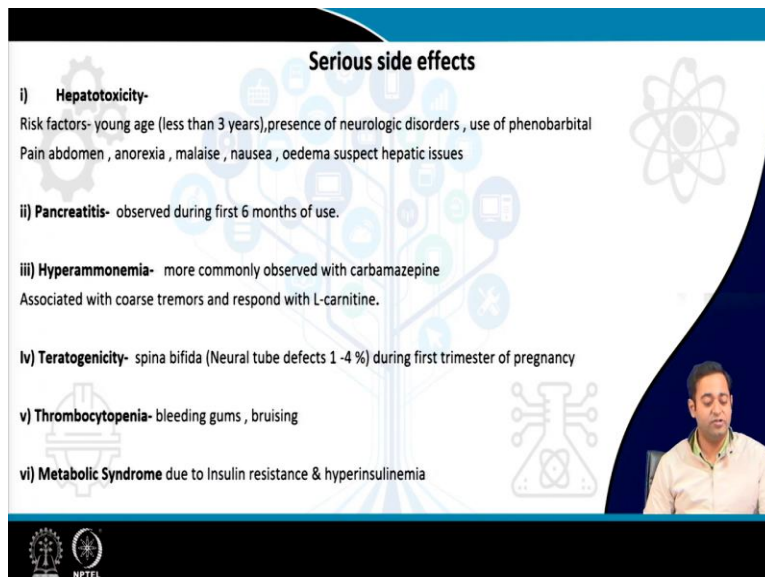
Valproate activates signals that promote neuroprotection and long-term plasticity, through

- a) Extracellular signal-regulated kinase (ERK)
- b) Cytoprotective protein B-cell lymphoma/leukemia-2 gene (BCL2), and GAP43

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The common side effects are in the form of nausea, vomiting, hair loss, weight gain, ataxia, tremors. The neuroprotective role is basically due to extracellular signal transduction cascades and cytoprotective protein B lymphoma that is BCL2 and glycogen AP43 proteins.

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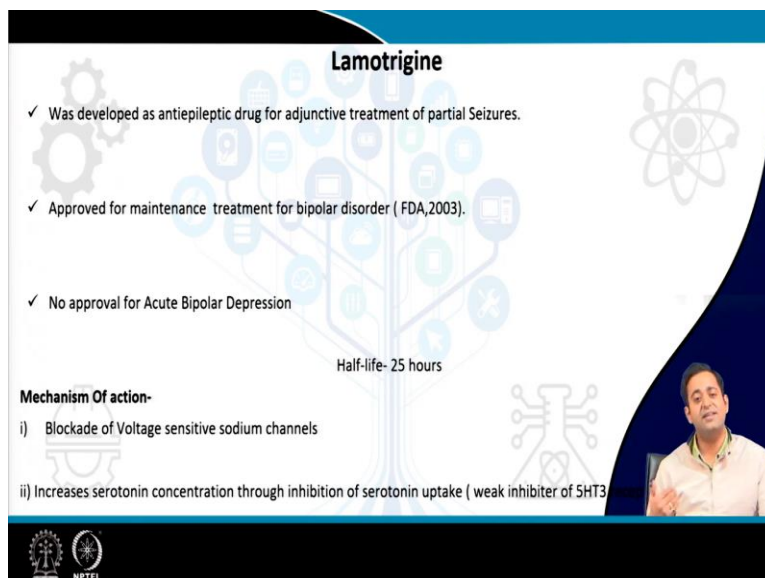
Serious side effects

- i) **Hepatotoxicity-**
Risk factors- young age (less than 3 years), presence of neurologic disorders, use of phenobarbital
Pain abdomen, anorexia, malaise, nausea, oedema suspect hepatic issues
- ii) **Pancreatitis-** observed during first 6 months of use.
- iii) **Hyperammonemia-** more commonly observed with carbamazepine
Associated with coarse tremors and respond with L-carnitine.
- iv) **Teratogenicity-** spina bifida (Neural tube defects 1-4 %) during first trimester of pregnancy
- v) **Thrombocytopenia-** bleeding gums, bruising
- vi) **Metabolic Syndrome** due to Insulin resistance & hyperinsulinemia

The slide features a background with a stylized tree of icons representing various medical and scientific concepts. A small inset video of a presenter is visible in the bottom right corner.

Serious side effects are in the form of hepatotoxicity, pancreatitis, hyperammonemia, teratogenicity that is (8:34) defects, thrombocytopenia leads to bleeding disorders, and metabolic syndrome due to his insulin resistance and hyperinsulinemia.

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Lamotrigine

- ✓ Was developed as antiepileptic drug for adjunctive treatment of partial Seizures.
- ✓ Approved for maintenance treatment for bipolar disorder (FDA, 2003).
- ✓ No approval for Acute Bipolar Depression

Half-life- 25 hours

Mechanism Of action-

- i) Blockade of Voltage sensitive sodium channels
- ii) Increases serotonin concentration through inhibition of serotonin uptake (weak inhibitor of 5HT3)

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Let us come to lamotrigine. So lamotrigine was antiepileptic drug for adjunctive treatment of partial seizures, it was developed due to this. Approved from maintenance of treatment of bipolar disorder that is FDA approved in the year 2003 and there is no approval as such for acute bipolar

depression although there are evidences for which lamotrigine is utilized and indicated in psychiatric (9:09) practice.

So half-life for the drug is 24 hours and the mechanism of action is block out of voltage sensitive sodium channels. And the second is increases serum constant serotonin concentrations through inhibition of serotonin reuptake.

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Side Effects
Common side effects- dizziness, somnolence, headache, diplopia, nausea are mild in nature.

Severe side effect
Rash- it can appear within first 4 months of starting the drug.
If the drug is titrated too fast with rapid hike in the doses, rashes can occur which may represent early manifestations of Steven-Johnson syndrome/Toxic Epidermal Necrolysis. (Incidence of Rash- 0.08%)
Children less than 16 years appear to be more susceptible to rash

Drug Interactions
i) Serious interaction with Valproate (Enzyme Inhibitor) which doubles the concentration of Lamotrigine.
ii) Increases concentration of lamotrigine when given along with sertraline, paroxetine).

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Side effects are in the form of dizziness, somnolence, headache, diplopia, at times nausea. The serious most serious side effect seen or associated with lamotrigine is the rash. So rash it appears within the first four months of starting the drug. And the drug is titrated too fast if you are hacking the drug too fast, it leads to rashes or more progressed severest form that is Steven Johnson's syndrome.

Children less than 16 years, they are more susceptible to this development of rashes. The drug interactions that should be most cautious for is valproate with which it doubles the concentration of lamotrigine, because valproate being an enzyme inhibitor and the ones are the accessorized paroxetine sertraline, they also increases the concentration of lamotrigine.

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Carbamazepine

- ✓ Initially developed as an Antidepressant
- ✓ Approved for use in trigeminal Neuralgia & Complex Partial Seizures

Half-life – 26 hours

(with chronic administration its half-life reduces to 12 hours due to its enzyme inducing properties, (autoinducer).

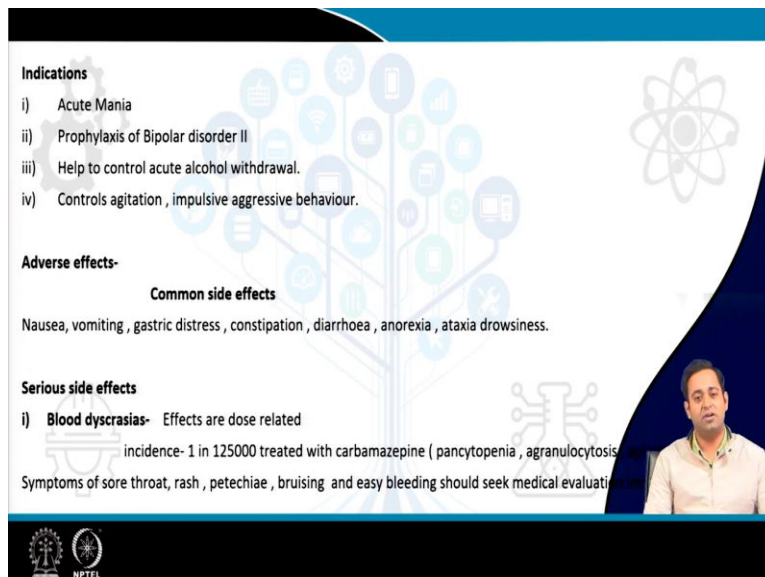
Mechanism Of Action-	Therapeutic dose range-	10-12mg/kg body weight
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- i) Binding of voltage dependant sodium channels in inactive state thereby prolonging their inactive state.
- ii) Reduces voltage dependant calcium channel activation.
- iii) Reduction of N methyl-D aspartate (NMDA) glutamate receptor channels.

Next is carbamazepine, the carbamazepine was initially developed as an antidepressant and later on it there was approval by FDA where it is being used for trigeminal neuralgia and complex partial seizures. So half-life for the drug is 26 hours but this half-life is decreased because of the continuous or chronic administration of the drug, reason being auto induction of the enzymes, enzyme inducer. Carbamazepine is a auto enzyme inducer.

What is the therapeutic dose range, that is it is calculated on the basis of 10 to 12 milligram per kg body weight. Mechanism of action is binding of voltage dependent sodium channels in inactive state thereby prolonging their inactive state. So it reduces the calcium channel activation and there is reduction of NMDA glutamate receptor channels.

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Indications

- i) Acute Mania
- ii) Prophylaxis of Bipolar disorder II
- iii) Help to control acute alcohol withdrawal.
- iv) Controls agitation , impulsive aggressive behaviour.

Adverse effects-

Common side effects

Nausea, vomiting , gastric distress , constipation , diarrhoea , anorexia , ataxia drowsiness.

Serious side effects

- i) **Blood dyscrasias-** Effects are dose related
incidence- 1 in 125000 treated with carbamazepine (pancytopenia , agranulocytosis)
Symptoms of sore throat, rash , petechiae , bruising and easy bleeding should seek medical evaluation

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What are the indications for carbamazepine? Acute mania, prophylaxis of bipolar disorder 2, it helps to control acute alcohol withdrawal, and it controls agitation, and impulsive aggressive behavior. That adverse effects are in the form of nausea, vomiting, gastric distress, constipation, diarrhea, ataxia at times drowsiness. The serious side effect is pancytopenia, agranulocytosis, and aplastic anemia.

So the symptoms of if at all the patients with a little bit symptoms of sore throat, rash, bruising, easy bleeding they should be seeking the evaluation from a physician immediately in order to stop the drug, because these are most notorious to cause pancytopenia, agranulocytosis, and aplastic anemia.

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ii) **Hepatitis-**

- a) Within the first few weeks of starting carbamazepine both hepatitis and increase in serum transaminases is seen.
- b) Persistent elevation of more than 3 times the upper limit indicates need to discontinue the drug.

iii) **Dermatologic effects-**

Rash is observed within 3 weeks of starting treatment

Some patients experience life threatening dermatologic syndromes namely , exfoliative dermatitis, stevens-johnson syndrome, toxic epidermal necrolysis.

Patients who are good responders to the drug can be continued with prednisolone 40 mg a day without having to stop carbamazepine.

iv) **Renal Effects-**

Carbamazepine activates vasopressin receptor function which results in a condition resembling the syndrome of inappropriate antidiuretic hormone characterized by hyponatremia(water intoxication).

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The next most serious side effect is hepatitis, it is it occurs within the first few weeks of starting the carbamazepine. So persistent elevation of more than three times the level of this leads to stoppage of the drug. Among dermatological effects, we have rashes which can be seen within the three weeks of starting the treatment.

And patients who are good responders to this drug can be continued with prednisolone 40 milligrams, if at all the patient does not respond to any other drug but carbamazepine. So they can be given with this, when the prednisolone that is corticosteroid is given along with the carbamazepine. Among renal effects you have a water intoxication where the patient results into syndrome of inappropriate antibiotic hormone.

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Maternal use of carbamazepine during pregnancy
Cleft palate, fingernail hypoplasia, microcephaly, spina bifida in infants

Carbamazepine treatment should be discontinued

- a) When WBC count < 3000/mm³
- b) Neutrophils below < 1500/mm³
- c) Serum iron < 150 mg/ml
- a) Platelet < 100,000 mm³
- b) Haemoglobin < 11g%

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There are various other contraindications where the carbamazepine can present with, that is if the child if the, if there is maternal use of carbamazepine during the pregnancy and the child might develop cleft palate, fingernail hypoplasia, microcephaly, and spina bifida. So there are some chances at the patient that the child might develop.

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Topiramate

Developed as an anticonvulsant useful in neurologic and psychiatric illness.

Indications-

- i) FDA approved for seizure as monotherapy or augmentation with other antiepileptics
- ii) Useful in prevention of migraine (FDA 2012).
- iii) Treatment of obesity, bulimia nervosa off label use
- iv) Useful in decreasing tremors.

NPTEL

Next is topiramate, topiramate is developed as an anticonvulsant and useful in neurological and psychiatric illnesses. What are the indications? It has the FDA approval for seizures as

monotherapy and also for augmentation with other various other antiepileptic drugs, it is useful for prevention of migraine also, and it has off-label use of treatment in obesity and bulimia nervosa off.

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Mechanism Of action

- a) Blocks voltage gated – sodium channels.
- b) Reduces membrane depolarization by AMPA/Kianate receptors.
- c) Enhances GABA (A) receptor activity

Most common adverse effects

- a) Paresthesias , weight loss , somnolence , anorexia , dizziness ,memory problems.
- a) Low serum bicarbonate – formation of renal stones.

Mechanism of action is it blocks, it acts by blocking the voltage sensitive sodium channels. And it reduces the membrane depolarization by Kianate or AMPA receptors. And it enhances the GABA receptor activity. Most common adverse effects are the paresthesias, weight losses, somnolence, anorexia, dizziness, memory problems. And there leads to formation of renal stones.

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REFERENCES

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- 2 Comprehensive Text book Of Psychiatry (Kaplan & Sadock)
- 3 Text book Of psychiatry (Tasman & Leiber mann)
4. Stephan stahl's psychopharmacology

CONCLUSIONS

-In this lecture we have discussed regarding mood stabilizer used in clinical practice for treating various psychiatric illness , its mechanism of action , indications & related side effects.

In this lecture we have discussed regarding the mood stabilizers that is lithium, valproate, lamotrigine, carbamazepine. Thank you.