


Basics of Mental Health and Clinical Psychiatry
Professor Dr. Sumit Kumar
Tata Main Hospital
Jamshedpur
Lecture 30
Emergency Psychiatry

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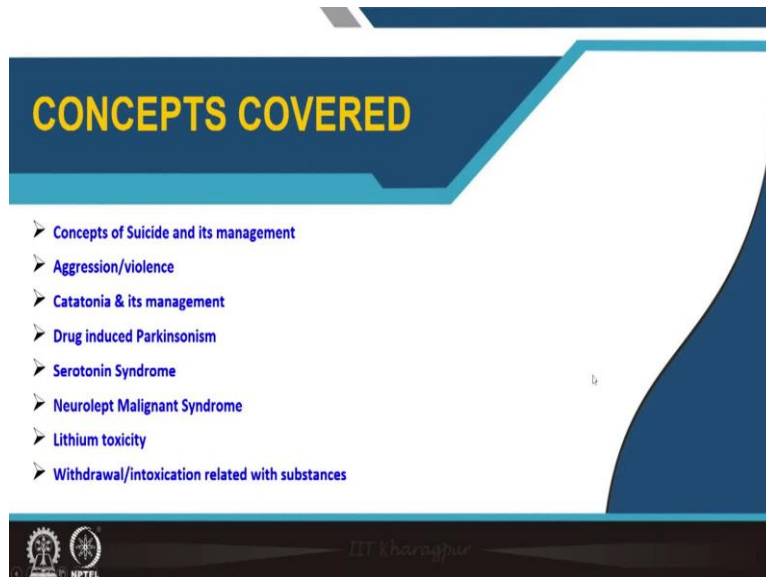


The slide features a blue header with two circular logos. Below the header, a blue banner reads "NPTEL ONLINE CERTIFICATION COURSES". The main content area is white and contains the following text:

Course Name Basics Of Mental Health & Clinical Psychiatry
Faculty Name Dr Sumit Kumar
Department Name Psychiatry
TATA MAIN HOSPITAL

Lecture 30 : Emergency Psychiatry

Navigation icons are visible at the bottom of the slide.



The slide has a blue header with the text "CONCEPTS COVERED" in yellow. Below the header, a list of topics is presented with blue arrow icons:

- Concepts of Suicide and its management
- Aggression/violence
- Catatonia & its management
- Drug induced Parkinsonism
- Serotonin Syndrome
- Neurolept Malignant Syndrome
- Lithium toxicity
- Withdrawal/intoxication related with substances

The footer of the slide includes the IIT Khargpur logo and the text "IIT Khargpur" and "NPTEL".

Hello, everyone. Let us start lecture number 30. That is emergency psychiatry. So what are the topics that we will be discussing? The concept of suicide, its management. Assessment of the violence, if any patient comes is aggressive and is very violent, catatonia and its management, drug induced Parkinsonism that is the psychiatric illnesses when the patient is under anti-psychotic drugs when they come in emergency with acute dystonia or NMS these kinds of conditions, serotonin syndrome, neurolept, malignant syndrome, lithium toxicity for

bipolar affective disorders, these are most commonly integrated drugs and withdrawal and intoxicated treated with certain substances.

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Emergency

- *It is a set of circumstances in which a catastrophic outcome is thought to be imminent and the resources available to understand and deal with the situation are unavailable at the time and place of the occurrence.*

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So, actually, what is an emergency? What do we know about an emergency? What is it? It is a set of circumstances in which a catastrophic outcome is thought to be imminent, it is about to happen it is inevitable and we do not have the resources available to mitigate the or to find a solution for that problem. And that is how it is actually being the patient is not able to manage very swiftly or smoothly in the emergency.

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Evolution of medical emergencies

Nature of medical emergency system has been previously running on a **horizontal level** wherein the duty doctor (level I) refers the patient to emergency medicine specialists level II) on site and then eventually to specialist level III & ultimately (Level IV) on a hierarchical fashion.

However in due course of time it was replaced by **vertical system** that defines a facility for emergency care capability for specific critically ill patient groups namely, trauma, burn, cardiac patients and behavioural etc.

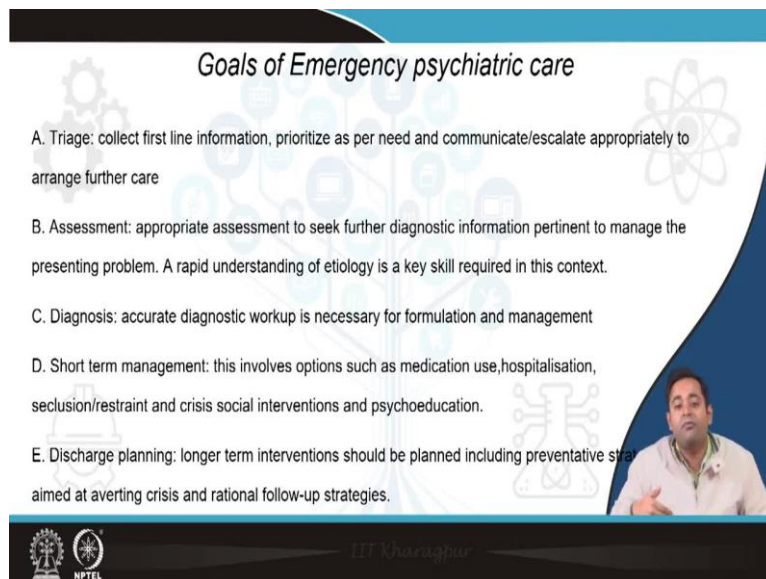
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So, how is that medical emergency, how has it evolved? The nature of medical emergency system has been previously on a horizontal level. Now, what is the horizontal level and what is the vertical level, medical emergency levels, the horizontal level is when you have a hierarchy where the specialists, the primary care physician in the primary healthcare levels, your first time the patient is being seen, they are being referred to CSCs and then from there, they are referred to tertiary care centers.

So, that is how there are level 1 level 2 level 3, depending upon the hierarchy the patient is being transferred or referred to higher centers, but in vertical system, you have those specialists there itself at the disposal of the patients in the emergency itself, you have all kinds of specialties like cardiologist, psychiatrist, surgeons, neurosurgeons, orthopedics, they all are there at your disposal to actually counter the problems which is present at the emergency.

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Goals of Emergency psychiatric care

- A. Triage: collect first line information, prioritize as per need and communicate/escalate appropriately to arrange further care
- B. Assessment: appropriate assessment to seek further diagnostic information pertinent to manage the presenting problem. A rapid understanding of etiology is a key skill required in this context.
- C. Diagnosis: accurate diagnostic workup is necessary for formulation and management
- D. Short term management: this involves options such as medication use, hospitalisation, seclusion/restraint and crisis social interventions and psychoeducation.
- E. Discharge planning: longer term interventions should be planned including preventative strategies aimed at averting crisis and rational follow-up strategies.

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So, what is the goal of those emergency psychiatric care? First is to try, to try this means try to like segregate the cases in like depending upon the importance of cases like red, yellow or green. So, green is where you give like most ambulatory patients, when you where you do not see very emergency kind of intervention, which is required, yellow is where Okay, there is somewhat in between like, not very urgency, not very urgently required intervention or which is like the patient can be given medication and asked to come follow up in the OPDs. So, after the triage, after the segregation of the patients, you have to go undergo the, patient has to undergo assessment.

So, in that assessment, the diagnosis has been done, brief workup of the patient is done and try to see whether this patient needs admission or this can be managed on the basis of OPD so, there at the emergency we have sometimes available psychologist or mental health professional who can undergo who can do the crisis intervention management strategies for the patient and the acute care for the patient is also given and it is actually seen whether patient will be managed in the inpatient that is in the ward itself or it can be followed up in OPD.

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Types of Psychiatric Emergencies

General Syndromes	Iatrogenic
1. Suicide	1. Drug induced parkinsonism
2. Agitation/Aggression	2. Neurolept malignant syndrome
3. Catatonia	3. Serotonin syndrome
4. Disinhibition	4. Lithium toxicity
5. Withdrawal/Intoxication	5. Priapism

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So, what are the types of psychiatric emergencies? Basically, divided into 2 class that is Iatrogenic that is drug induced emergencies and the general that is suicide, aggression, agitation, catatonia and withdrawal substances or intoxicated substances.

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Suicide

WHO-states that

"Suicide is an act, deliberately initiated and performed by a person in the full knowledge or expectation of its outcome"

Deliberate self harm- Wilful self inflicting of painful, destructive, or injurious acts without intent to die.

Suicidal Intent – subjective expectation and desire for a self- destructive act to end in death.

Lethality of Suicidal behaviour-Objective danger to life associated with a suicide method or action

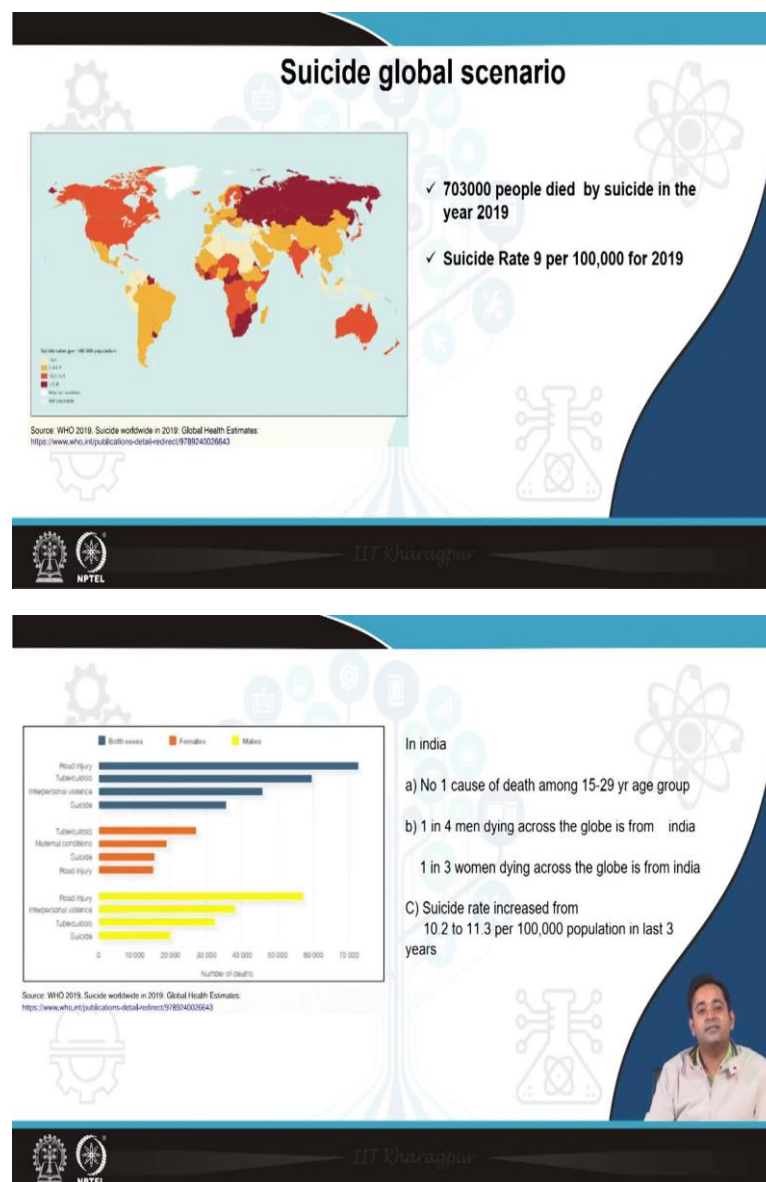
So, Let us start with suicide first. What is it suicide? Suicide is an act deliberately initiated and performed by a person in the full knowledge or expectation of his outcome. So first there is a conceptualization of the act that is that the patient is going to undergo then there is understanding, if this is the way I am going to act and this is going to be the deliberate outcome. So, that is ultimately it is a behavior. So, there is conceptualization of a problem, there is understanding and then there is an execution, 3 most important entities that is, that the patient undergoes in order to commit suicide.

So, it is a very deliberate, a very impulsive kind of act. Sometimes it is a very foolproof plan that the patient wants to end his life depending upon his circumstances in their life. So, what is deliberate self-harm, suicidal intent and lethality, some important terms related to suicide,

deliberate self harm is when the patient is there is no intent to die, but there is self inflicting pain and destructive or injurious acts done to himself or herself. The intent is when there is a when there is a desire for self destruction, which can lead to death of the patient.

And lethality is the method of associated the method of committing suicide the outcome of the method which the patient tries to undergo, where he or she very well knows that this is the method if I try to execute there is more than 100 percent chance of ending my life.

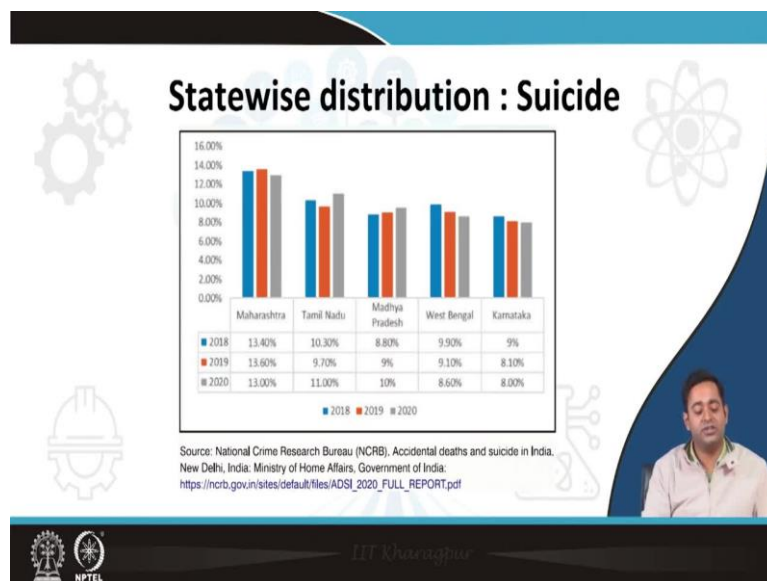
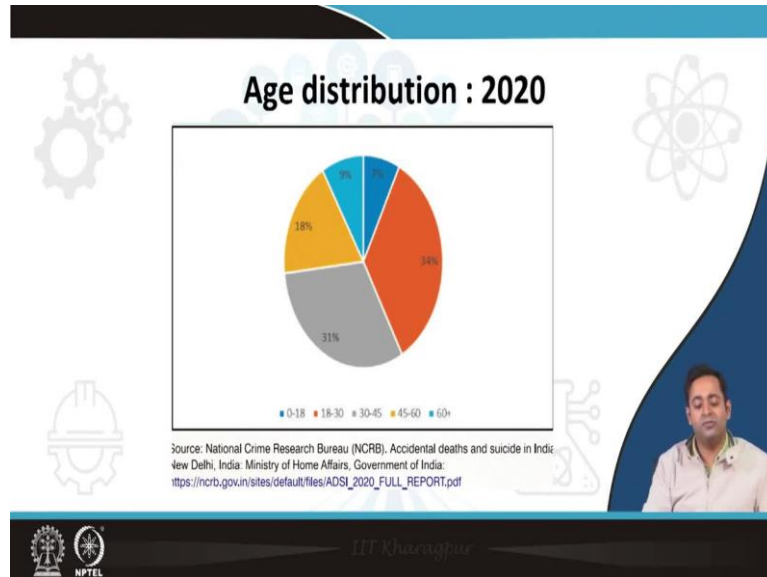
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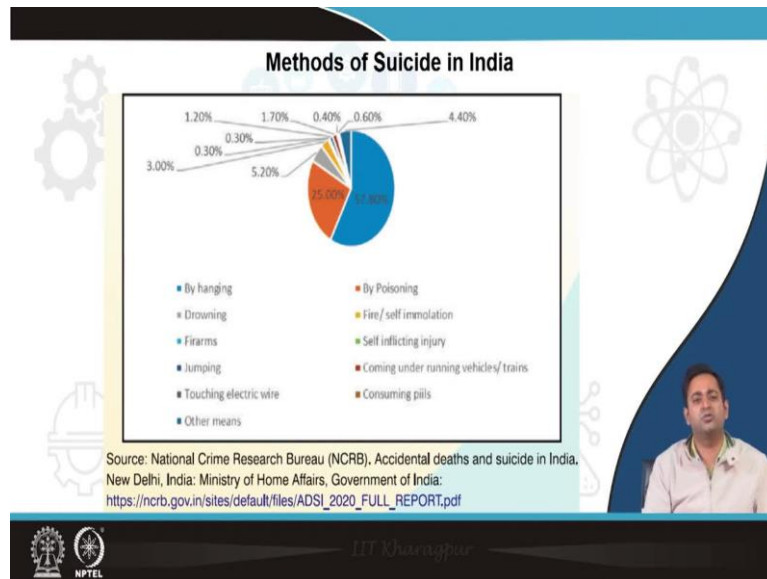


So if you see the global scenario of suicide, 703000 people died by suicide in the year 2019. And suicide rate is 9 per 100000 for 2019 for the year 2019. These are the case reports from Global Health estimates. In India, it is the number one cause, suicide is the number one cause of death among 15 to 29 years age group and 1 in 4 of men and 1 in 3 of women. If you

compare this to statistics across the globe, it is 1 in 4 of men and 1 in 3 of women that they are committing suicide and the suicide rate if you compare from last 3 years, it has increased from 10.2 to 11.3 per lakh, per 100000 population.

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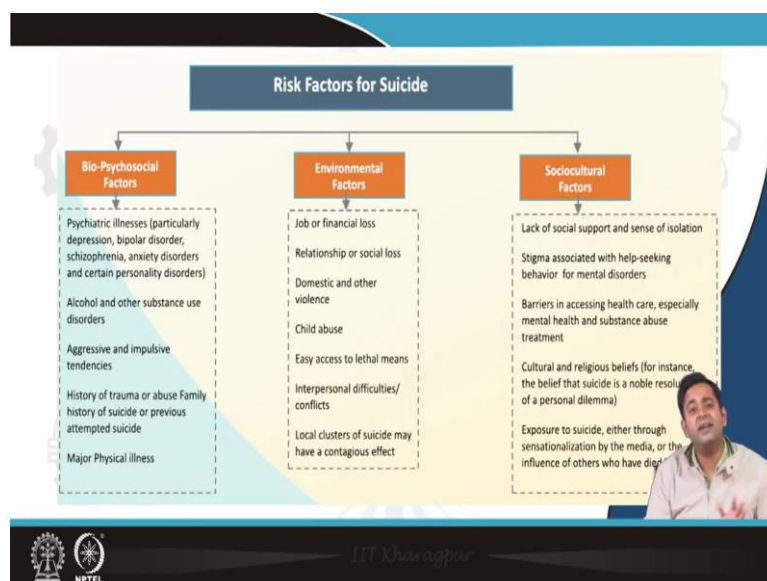




Age distribution as we have seen previously, it 15 to 29 years of age group. The data reveals it is 34 percent belong to 18 to 30 years of age group and a 31 percent for 30 to 45 years of age group, state wise distribution in India, the Maharashtra state tops the list of committing suicide peoples committing suicide.

Among the methods of suicide, which is taken by the patient, hanging is the most common means of committing suicide, followed by poisoning, then you have drowning and firearms, touching electric wires, going under the railway tracks, self inflicted injury, those are the methods by which patient tries to commit suicide.

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What are the risk factors of suicide basically divided into 3 class, bio psychosocial factors, environmental factors and socio cultural factors. If you look at the bio psychosocial factors,

these are the patients who are suffering from chronic illnesses, they can be medical illnesses or psychiatric illnesses. So medical illnesses, you have all sorts of cancer, carcinomas, orthopedic, tumors, your long standing surgeries, cranial surgeries, and in case of like non communicable diseases, you have long standing diabetes where the patient lands up in taking insulin from last 10 years, they are in a very pathetic kind of state.

And the conditions or the states which act which further adds to the misery of the patient is when they become bedridden, crippled, and they are not, the mobility is lost for the patient. In those kinds of circumstances patient actually tries to commit suicide, thinking of this miserable life of himself or herself. So you have all sorts of psychiatric illnesses, bipolar schizophrenia, you are the treatment for the patient, it is there from last 10 years, 20 years and they are not coming out of the illness, the trajectory of the prognosis is very bad, they are not feeling that they should be able to recover from the present ongoing illnesses.

Then you have substance abuse, the patient who are undergoing or who are under the effect of this notorious substances like nicotine, alcohol, cocaine, MDMAs, cannabis. They are abusing it from last 10 years, 20 years and howsoever hard they tried they are not able to quit this habit of this and so they become like frustrated and they try to impulsively commit suicide by escaping from the situation that now I will not have to deal with this situation.

So you have environmental factors, what are the common features under environmental factors you have relationship issues between the parents you have marital conflict or you have conflict with your family members, relatives, even your friends.

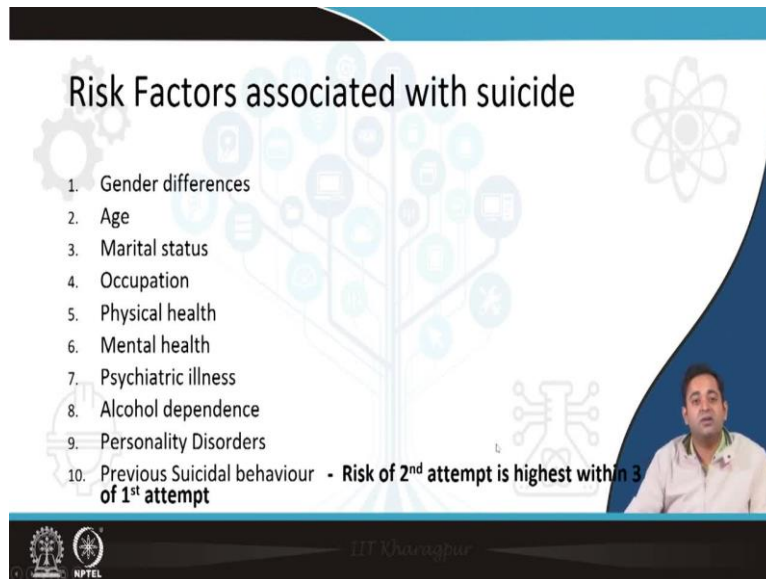

So, all of these can actually culminate into committing suicide, you have in case of childhood abuse, you have physical, sexual, or in case of elderly, you have economical abuse, physical abuse.

So, these are all those factors interpersonal difficulties among maybe family members or friends, families, the place where you live, the address, the locality where you habitat is not conducive enough, everybody is like trying to segregate you try to ostracize you banned from society. Because you are not able to mingle because of the undergoing substances, you are undergoing some treatment for psychiatric illnesses, so, they are not allowed the stigma associated with those issues. They are not allowing you to come and mingle and try to intervene with this society, the integration of the society is not proper, all of these factors, they culminate into suicide.

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Risk Factors associated with suicide

1. Gender differences
2. Age
3. Marital status
4. Occupation
5. Physical health
6. Mental health
7. Psychiatric illness
8. Alcohol dependence
9. Personality Disorders
10. Previous Suicidal behaviour - Risk of 2nd attempt is highest within 3 months of 1st attempt



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Etiology

- I. Sociobiological
 - a) Egoistic
 - b) Altruistic
 - c) Anomic
- II. Psychological
- III. Biological
- IV. Genetic



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The most important thing is the risk of second attempt is highest within the 3 months of first attempt of suicide. What are the Etiological factors, the first and most important is genetic. Now, there are studies and evidences available, where the first relatives like if the parents mother or the father, if they have committed suicide, so, it actually predisposes the patient the next generation that there is high genetic loading in the gene, which predisposes the patient to commit suicide.

So, that does not mean that if your father or your mother has committed suicide in the past and you will be committing suicide in the future, it gives up it means you are being predisposed there is a gene which depending upon the circumstances and environmental

factors can actually precipitate or impulsively patient can commit suicide by trying to escape out from the situation if he or she is not able to manage it.

Now, those sociological theories, Emily Durkin was the person who was actually working on this and he tried to classify this sociological theories into 3 types that is egoistic, altruistic and animistic. The atomistic one is, as you know, the patient tries to like give up all these worldly pleasures and just to satisfy the ego of others, like the say for example, the border, the person who is there at the border, the army persons, they sacrifice their lives for the sake of their entire nation.

If you come with like, egoistic, egoistic is where the person does not try to socially integrate in the society and that is how the literature and the evidence says it says, it says that those who are not integrating into society are more predisposed to commit suicide, like the married members of the family, they are more protective of not committing suicide, the ones who have children, they are more protective of not committing suicide, because there is the sense of belongingness with the family, which actually tries to or tries to inhibit the person from committing suicide because they think they have someone who he or she has to look after they have to think for the future.

A biological factors, Serotonin is a neurotransmitter which is responsible for the mood of the person. So, there are some evidences which tells that the metabolite of serotonin that is a 5 hydroxy indole acetic acid, it is decreased in the CSF, the metabolites, the duration factors of the serotonin, they are decreased in case of patients suffering from depression, who ultimately land up in committing suicide. So, there are evidences available so there is Gene also, which is predispose in the patients who are committing suicide TPH gene tryptophan hydroxylase gene is present in the patient. The most important factor is the L allele, which is present in the patients who are committing suicide.

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Assessment of suicide

Scales commonly used for assessment of suicide

- Becks Suicide Intent Scale – Developed by Aaron T Beck of University of Pennsylvania.
- Columbia Suicide Severity rating scale- developed by John Mann of New York State Psychiatric Institute

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Now, how are we supposed to assess the suicide? It is basically done by commonly 2 scales, what is Beck Suicide Intense Scale and the Columbia Suicide Severity Rating Scales, both these scales are done like they are performed to assess the intent, lethality, the how, what is the method of committing suicide is it like foolproof it is like they have the persons who have actually tried to commit suicide. And they have tried it and not succeeded in the past.

So there has been some previous attempts, or they are committing suicide under the effect of substances like alcohol, nicotine or opium or cocaine or anything else. So this case, they actually try to assess the patient who might commit suicide in the following headings.

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Goals to reduce suicide

- ✓ Promote awareness that suicide is a public health problem that is preventable.
- ✓ Develop & implement strategies to reduce stigma associated with a consumer of mental health , substance abuse & suicide prevention services.
- ✓ Promote efforts to reduce access to lethal & methods of self harm.
- ✓ Develop & promote effective clinical & professional practices.
- ✓ Improve reporting & portrayals of suicidal behaviour , mental illness & substance abuse in the entertainment news media
- ✓ Promote & support research on suicide & suicide prevention.
- ✓ Improve & expand surveillance sysytems

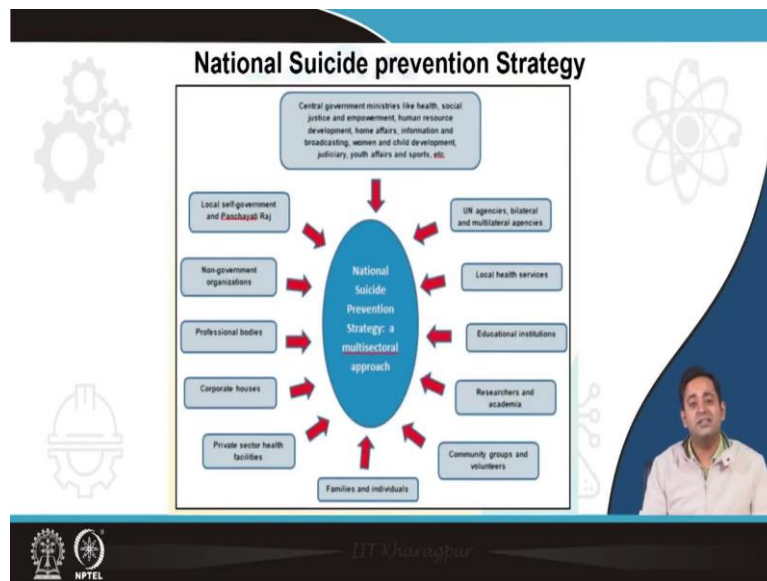
The slide features a background with a stylized tree of icons representing various fields of study. A small inset video of a speaker is visible in the bottom right corner. The NPTEL logo and 'IIT Khargapur' text are at the bottom.

So, what should be the goal? The ultimate goal is to prevent this nuisance, first and foremost is foremost is to promote the awareness that suicide is a public health problem, and it is actually preventable, develop implement strategies to reduce the stigma associated with those 3 important consumer services, that is ones who were using substances, the ones who are undergoing mental treatment, the treatment of various psychiatric illnesses, and the ones who might be committing suicide, which have been like these patients who are undergoing treatment and they want to prevent their execution of suicide. So they seek for these opportunities this services. So we should try to implement strategies to reduce stigma, so that these patients should come up and seek help.

Promote efforts to reduce access to lethal and methods of self harm, that is firearms or poisonous substances, or like the tablets which are procured by the patients should not be given without a valid prescription. Develop and promote effective clinical and professional practices. Improve reporting and portrayals of suicidal behavior. This is a very important point here. Because the sensationalization of the reporters in various public media news, or internet, it actually gives a kind of courage that is the way we can actually escape out from the problem so that people can remember us in future. So there should be a responsible reporting of this kind of portrayals in the media.

Promote and support research on suicide and suicide prevention. And we should really expand the surveillance system, the various epidemiological surveys as to how the persons who are living in the communities how are they coping with the normal day to day life problems.

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Now, there is a national suicide prevention strategy where you have a holistic plan from all sorts of agencies like government agencies, community groups, NGOs, local self government, non government organization that is NGOs, professional bodies, so everybody should act in collaboration, to actually try to inhibit this nuisance, and that will help in further prevention of the suicide.

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Aggression/Violence

Assessing & predicting violent behaviour

I Signs of recent violence

- a) Very recent acts of violence, including property violence
- b) Verbal or physical threats
- c) Carrying weapons or other objects that may be used as weapons eg (forks , ash trays)
- d) Progressive psychomotor agitation
- e) Alcohol or drug intoxication / Paranoid features in psychotic patients
- f) Command auditory hallucinations / Organic mental disorders global or frontal lobe findings
- g) Catatonic excitement
- h) Patients with mania
- i) Patients with agitated depression / Personality disorders prone to rage , violence

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What is aggression and violence? So how are we going to assess this kind of behavior in the emergency, they had some lookouts, certain points that the person should be looking for the ones who are assessing the patient at the emergency. Acts of recent violence, verbal or physical threats, like I am going to harm you this is the reason for which I am here.

Progressive psychomotor agitation, if the patient is under some kind of substances like alcohol, nicotine, or all kinds of things, some paranoid features if the patient is suffering from psychiatric illnesses, delusions, hallucinations, that can this can further provoke and gives rise to this manifestation of agitation violence or aggression. This can also be under due to command auditory hallucinations, where the voices are actually commanding you to behave in a certain kind of manner.

Catatonic excitement, a patient with mania or who are undergoing agitated depression. These are the situations where the patient might behave or create violence or aggressive patient can be aggressive.

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Assessing risk of violence

- a) Consider violent ideation, wish, intention, plan, availability of means, implementation of plan.
- b) Consider age, gender, socioeconomic status, social support
- c) Consider past history violence, nonviolence, anti social acts, impulse dyscontrol (gambling, substance abuse, suicide, self injury)
- d) Consider over stressors like marital conflict or death of near ones

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Assessing of the risk of violence, consider violent ideation, wishes intent, if the patient is like, he is not verbally expressing it out, but there is an intent which is which has to be observed by the non verbal behaviors, the body language of the patient. Consider the age, gender, socioeconomic status, status, social support, past history of what has where the patient has committed this kind of aggressive behaviors in the past, and the antisocial personality disorders, where the patient are supposed to act in a certain kind of way. It is not that it is very deliberate that this will be acting, but because they are predisposed to act, because of the personality disorder that they are inhabiting.

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Under section 94 Of MHA 2017

- i) Any medical treatment may be provided subject to
 - a) Informed Consent of the NR
 - b) If it is of immediate danger to prevent danger to self or others
 - c) The person causing serious damage to property belonging to self or to others.
- ii) Treatment referred shall be limited 72 hrs only

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Admission under 85/86 if **voluntarily** willing to get admitted
Under section 89 with Nominated Representative for **involuntary admission**.

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So, under Section 94 of MHA, that is Mental Health Act 2017 any medical treatment may be provided to this kind of patients subject to informed consent of the nominated representative. So, this kind of patients if they are accompanied by a person or family or a relative or a friend, so, if he or she happens to come forward and seek for help, it is because of their informed consent, they are concerned that this kind of patients can get treatment, it does an immediate danger to prevent this to self and to others and the person causes serious damage to other belongings to sell for others.

So, these are the criteria's where the section 94 can be implicated. So, this should be done under 72 hours. And if the patient is voluntarily getting, wanting to get treated himself can be admitted under 85 86 of MHA. Or if the patient does not have the capacity, then obviously with the help of a nominated representative, this kind of patients can be admitted under Section 89 of the Mental Health Act.

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Catatonia

As defined by DSM-V

Presence of 3 or more of the following

a) Catalepsy	b) Waxy flexibility
c) Stupor	d) Agitation
e) Mutism	f) Negativism
g) Posturing	h) Mannerism
i) Stereotypies	j) Grimacing
k) Echolalia	l) Echopraxia

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So, what is catatonia? Catatonia is a neuro psychiatric conditions. It is basically a triad of mutism posturing, and you are all sorts of catalepsy, basically 3 types of things mutism, negativism and posturing 3 are the triad which is basically looked after for diagnosing catatonia presents of 3 or more.

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Stupor- No Psychomotor activity and no reactivity to the activity

Automatic obedience- Subject does whatever is asked of him /her despite not to do.

Ambitendence- Patient makes alternating co-operation and opposition

Aversion- Turns away from the examiner when addressed

Echolalia- Subject repeats the words & phrases

Echopraxia- Subjects imitates the actions/ movements of the examiner

Repetition of response after the withdrawal of the stimulus.


Mitmachen-Body can be put into any posture despite instructions to resist


Mitgehen-Extreme form of mitmachen when slightest pressure results in movement in any direction


Waxy flexibility- Smooth resistant muscle tone is felt to initiate movement.

Geganzhalten- A form of negativism in which resistance to passive movement increases with force exerted.


Catalepsy-If a subject's body posture is left in an awkward posture and left, the posture is held for a period before slowly asking the patient to relax




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Waxy Flexibility



Psychological Pillow - Catalepsy



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Now, there are various signs and symptoms when the patient presents in the emergency. Most important is stupor, stupor is actually accompanying 2 components that is immobility and mutism where the patient does not move is not able to move around and he is not able to speak so there is mutism and as well as immobility, automatic obedience is when the subject is asked to do but when the subject is asked to do something, but there is also an instruction that you are not supposed to perform like this, but the patient howsoever acts deliberately.

So what is MB tendency, so MB tendency is when the patient tries to alternatively cooperate and go back and not cooperate at the same time simultaneously. So it can be exemplified in the form of hand shaking where the examiner tries to shake hands with the patient. So patient

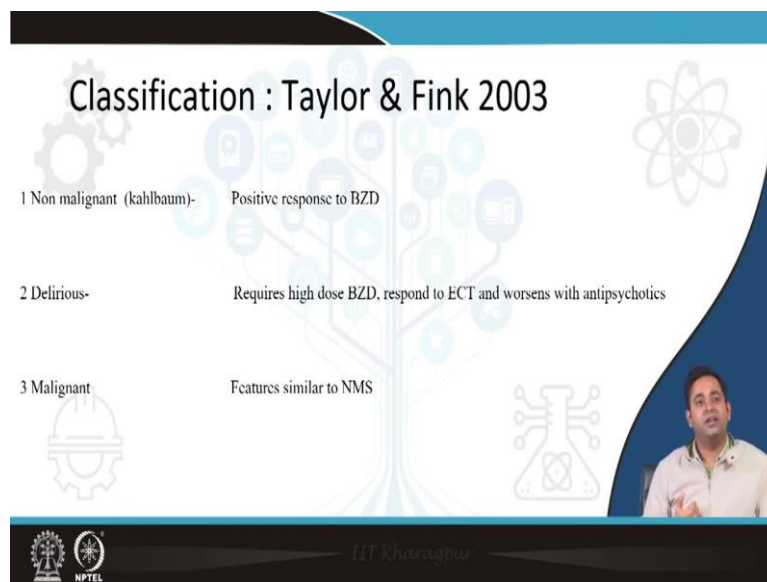
tries to move his hands forward and at the same time moved his hand backwards. So there is an opposition and as well as cooperation on the part of patient.

So there is Echolalia Echopraxia, Echolalia Echopraxia is when Echopraxia is when the patient tries to imitate the actions of the examiner or the one who is trying to assess and Echolalia is when the patient is trying to repeat the words of what the examiner is asking or telling.

There is Mitmachen and Mitgehen, posturing that is catalepsy, all those symptoms, then you have a Gagenhaltan, this is the kind of negativism, regulative rigidity, it is a kind of negativism where the activate where in which there is active resistance and this passive this resistance increases when you try to execute a movement, so there is the opposite resistance.

So catalepsy is when a subjects body posture is left in an awkward position, and the position is held for a brief period of time before slowly relaxing, despite asking the patient to relax. So we have a description of that is waxy flexibility and psychological pillow. So the patient is in a kind of uncomfortable position here and he tries to maintain this kind of position for minutes to ask together.

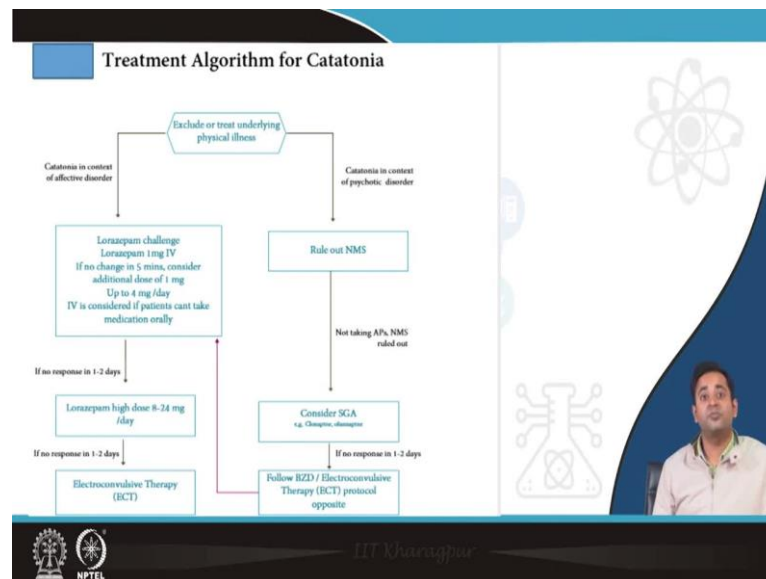
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That is one of the classification. Classification is basically of 3 basic types that is non malignant delirious and malignant. So normally, it is responding to benzodiazepines. Delirious type is it requires high doses of benzodiazepine, and as well as it responds to ECT, but it worsens to antipsychotics. Malignant catatonia is somewhat having features similar to NMS it is not exactly but somewhat features similar to NMS.

The pathophysiology of catatonia it is all proposed mechanism is that you have alternative the dysfunctional levels of glutamate, dopamine and GABA and there is decreased GABA receptor activity in the orbitofrontal part of the brain frontal cortex and the parietal lobe of the your cortical area and you have increased glutamate activity which is giving rise to this kind of affective motor or mood symptoms.

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So how do you treat catatonia? The drug of, I will not say see the drug of choice the first and foremost treatment for catatonia is benzodiazepines. Why because the neurotransmitters is GABA, glutamate and dopamine, it is there disturbances in the various lobes, which is causing this kind of many manifestations. So, if you go back and try to see the lectures of schizophrenia, where I have discussed the GABA levels each and this glutamate this glutamate, GABA and dopamine they are under feedback inhibition kind of system. So, GABA tries to negatively give the dopamine.

The dopamine levels and glutamate levels they are under feedback inhibition. So if you give the, if you increase the GABA levels by giving benzodiazepines this kind of scattering symptoms can get negative.

So first, we try to give oral benzodiazepines specifically Lorazepam. If there is no improvement in the symptoms, we try to give intramuscular Lorazepam if there is no symptoms, no relief of the symptoms, we try to increase the dosage of 2 to 4 milligrams for the 8 milligrams and 16 milligrams and give rise to if and if there is no relief of the

symptoms. We try to give the intravenous injections. If there is no improvement with intravenous injections at all ECT is indicated.

So we have actually deleted this kind of treatment there is a delineation of the treatment where the psychosis is involved with catatonia or if there is a mood disorder, which is involved in Catriona. So if there is a psychosis, atypical antipsychotics can also be tried and FDA is suggest the 2 most important antipsychotics which is used as aripiprazole, and risperidone with Proserpine, which has a good response rate.

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Iatrogenic Emergencies

Psychiatry Armamentarium consists of more than 50-60 molecules at our disposal.

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But We majorly rely on 4 classes of psychotropics to treat most of our patients in Opd.

These are mainly:-

↓

i) Mood Stabilisers ii) Antipsychotics iii) Antidepressants iv) Benzodiazepines

iatrogenic/ Psychotropic induced emergencies

Antipsychotics

1. Acute dystonia
2. Akathasia
3. Neurolept malignant syndrome
4. Antipsychotic Overdose

Antidepressants


1. Serotonin withdrawal syndrome
2. Serotonin syndrome
3. Priapism
4. Anticholinergic Syndrome
5. Overdose due to ssri's tca's, snri's

Mood stabilisers

1. Lithium toxicity
2. Valproate toxicity

Benzodiazepines

1. Intoxication
2. withdrawal



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Let us come to Iatrogenic emergencies, the drug and use cases. So here in psychiatry, we have more than 50 to 60 molecules at our disposal, from which we try to treat the patients and we heavily rely on these 4 classes. What are these 4 classes? These are basically mood stabilizers, antipsychotics, antidepressants, benzodiazepines.

So among antipsychotics, we have acute dystonia, Akathasia, Neurolept malignant syndrome and Antipsychotic overdoses. Antidepressants, we have SSRI withdrawal syndromes, serotonin syndromes, priapism, anticholinergic syndrome and overdose due to SSRIs SNRIs, or TCA. Mood stabilizers you have lithium toxicity and Valproate toxicity, and lastly, benzodiazepines intoxication or withdrawal phases.

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Antipsychotics
Related emergencies



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Dystonia is defined by involuntary maintained contraction of agonist and antagonist muscles yielding abnormal posturing, twisting and repetitive movements, or tremulous and can be initiated or worsened by attempted movement.

It assumes a pattern of

- overextension or over-flexion of the hand
- Inversion of the foot
- Lateral flexion or retroflexion of the head,
- Torsion of the spine with arching and twisting of the back
- Forceful closure of the eyes, or a fixed grimace.

It may come to an end when the body is in action and during sleep.

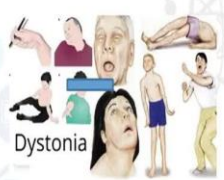


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So let us start with antipsychotic related emergencies. Dystonia is the first thing that we encounter in an emergency where the patients they come with over extension or over flexion of the hand inversion of the foot lateral flexion retro friction force closure of the eyes will lead to a curb light Gary crisis, but there is all kinds of symptoms they decreases if the patient is in action, or they are sleeping.


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Presentation at Emergency



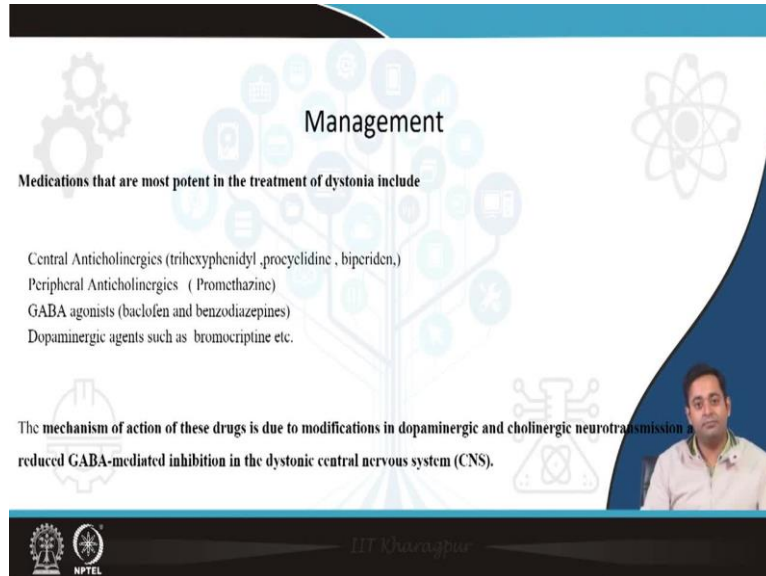
Dystonia

- Oro-mandibular dystonia can manifest as night-time bruxism (jaw spasms producing forceful jaw closure) and may follow dental procedures, oro-mandibular-facial trauma, or temporomandibular joint dysfunction.
- Spasmodic dysphonia can commence as a strained voice. Despite the name dystonia, the tone of affected extremities at rest is often normal at the onset.
- In cervical dystonia, head tremor sometimes decreases with the head held in a specific position, and displacement from that position, such as turning the head to one side, may worsen it.



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Dystonia- Acute emergency where there is difficulty breathing can be seen during Opoid withdrawal or increase in Antipsychotic dosage.



Management

Medications that are most potent in the treatment of dystonia include

- Central Anticholinergics (trihexyphenidyl, procyclidine, biperiden,)
- Peripheral Anticholinergics (Promethazine)
- GABA agonists (baclofen and benzodiazepines)
- Dopaminergic agents such as bromocriptine etc.

The mechanism of action of these drugs is due to modifications in dopaminergic and cholinergic neurotransmission and reduced GABA-mediated inhibition in the dystonic central nervous system (CNS).

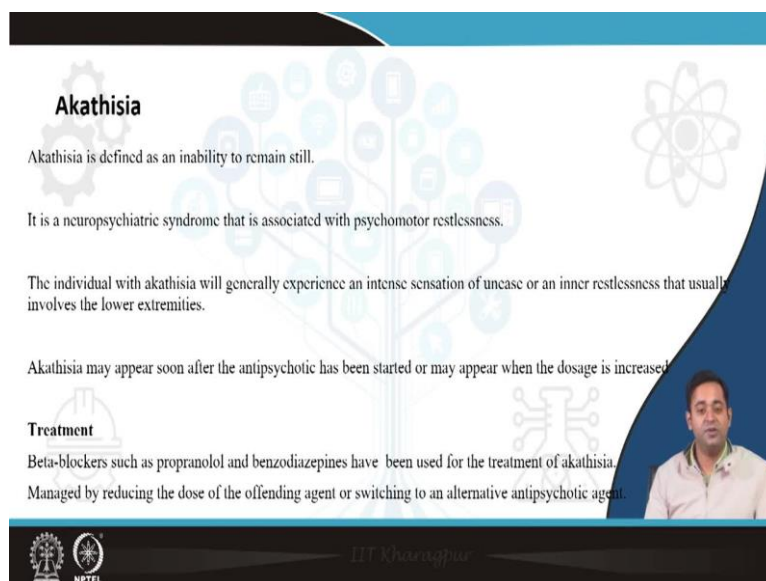
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So there are as I have told you, they can be various presentations of this dystonia, cervical dystonia as laryngeal dystonia, spasmodic dystonias, oromandibular dystonia. How do we manage it? Managing it by central anticholinergic that is trihexyphenidyl, biperiden, procyclidine, Peripheral anticholinergics this is promethazine, GABA agonists baclofen and benzodiazepines are also given to treat this kind of conditions.

And lastly we have dopaminergic agents that is bromocriptine. So do not mechanism of action of these drugs is due to modifications in dopaminergic and cholinergic neurotransmission and reduced GABA mediated inhibition in a dystonic central nervous system.

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Akathisia

Akathisia is defined as an inability to remain still.

It is a neuropsychiatric syndrome that is associated with psychomotor restlessness.

The individual with akathisia will generally experience an intense sensation of unease or an inner restlessness that usually involves the lower extremities.

Akathisia may appear soon after the antipsychotic has been started or may appear when the dosage is increased.

Treatment

Beta-blockers such as propranolol and benzodiazepines have been used for the treatment of akathisia.

Managed by reducing the dose of the offending agent or switching to an alternative antipsychotic agent.

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Drug induced Parkinsonism

Tremors - Drugs notorious to cause are selective serotonin (and norepinephrine) reuptake inhibitors (SSRIs/SNRIs) amitriptyline, lithium, valproate, β -adrenoceptor agonists, dopamine receptor antagonists etc and are managed by reducing the dose of offending agent and with the help of β blockers.

Dystonia - Develops within hours to days of initiation. Presents as muscle spasm, stiffness, oculogyric crisis)

Akathisia - (Develops within days to months of use of haloperidol - characterized by restlessness.)

(Tardive Dyskinesia - (Develops after years, presents as a chore, especially orofacial region)
managed by tetrabenazine VMAT2 receptor blocker

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What is Akathisia? It is a subjective restlessness on the part of patient and compelled desire to move out. So individuals with Akathisia they generally experience this intense kind of sense of uneasiness and inner restlessness, most and preferentially involving the lower extremities. The treatment is by beta blockers preferentially propranolol and by the benzodiazepines. So, what are the drug induced Parkinsonism that can actually present in the emergencies?

These are tremors the most notorious ones to cause are the SSRIs SNRIs lithium valproate among psychiatric psychotropics, which are given to the patients lithium valproate, beta adrenoceptor agonist, dopamine receptor antagonist. And they all are managed by reducing the dose or with the help of beta blockers, dystonia we have already discussed. So what is tardive dyskinesia? Tardive dyskinesia is developed some months and years of the treatment of anti psychotic usage. Specifically, they start with the orofacial region and they are managed by tetrabenazine. That is vesicular monoamine to receptor blockade.



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Neurolept Malignant Syndrome

- ❑ It affects 0.01 %- 3.2 % of patients taking neuroleptic medication.
- ❑ M:F - 2:1
- ❑ Young population due to involvement of antipsychotics in early age group.

Pathophysiology

1. Sudden reduction in central dopaminergic activity due to a D2 receptor blockade
2. Abrupt withdrawal of D2 receptor stimulation.
3. Develops NMS within hours or days after exposure to a causative drug, with most exhibiting symptoms within 2 weeks and nearly all within 30 days
4. Mental status changes, Fever, Muscle rigidity, and Autonomic Instability.



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Drugs causing NMS



Medications

Antipsychotics

- Haloperidol
- Fluphenazine
- Chlorpromazine
- Risperidone
- Clozapine
- Amisulpride

Antiemetics

- Domperidone
- Metoclopramide
- Prochlorperazine



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Let us come to be Neuroleptic Malignant syndrome. So, what is Neuroleptic Malignant syndrome? It is sudden reduction in the dopamine levels due to D 2 receptor blockade and there is or there can be abrupt withdrawal of the due to SEPTA stimulation. It develops within hours or days after exposure to the notorious drugs, most commonly typical antipsychotics.

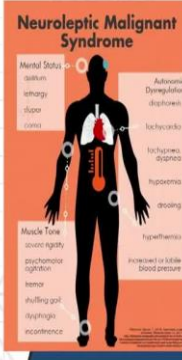
And there are features in the form of mental state changes that is confusion, irritability, agitation, that can be fever, muscle rigidity, and autonomic instability that is blacky cardio, postural giddiness, all those kinds of features are present when the patient is presenting in the casualty with NMS. What are the drugs that causes NMS, these are these can be antipsychotics antiemetics among antipsychotics, not most materials to call other typical ones

that is haloperidol chlorpromazine and among antiemetics we have Metoclopramide or Domperidone.

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Diagnosis of NMS: As per (DSM)

A.	Development of severe muscle rigidity and elevated temperature associated with the use of neuroleptic medication.
B.	Two (or more) of the following: (1) Diaphoresis (2) Dysphagia (3) Tremor (4) Incontinence (5) Changes in level of consciousness ranging from confusion to coma (6) Mutism (7) Tachycardia (8) Elevated or labile blood pressure (9) Leucocytosis (10) Laboratory evidence of muscle injury (eg, elevated CPK)
C.	The symptoms in criteria A and B are not due to another substance or a neurological or other general medical condition.
D.	The symptoms in criteria A and B are not better accounted for by a mental disorder



Neuroleptic Malignant Syndrome

Mental Status: delirium, stupor, coma

Autonomic Dysregulation: diaphoresis, tachycardia, tachypnea, dyspnea, hypoxemia, bradycardia, hypotension

Muscle Tone: muscle rigidity, psychomotor agitation, tremor, shivering, goiter, dysphagia, incontinence

Hypertension: increased or labile blood pressure

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What is the diagnosis of NMS, it is development of severe muscle rigidity, or elevated temperature associated with the use of neuroleptic medication. And 2 of the following features can be present with a lab diagnosis of increased creatine kinase levels, which leads to muscle injury ultimately, increased those muscle injury leads to acute renal failure, those myoglobinuria they occur and ultimately leading to acute renal failure.

So symptoms in criteria that is those muscle rigidity or a temperature they can be present. But these all conditions should not be due to a general medical condition or due to any some kind of substance which the patient might be abusing it.

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Treatment

- NMS is a Neurologic Emergency delay in diagnosis and treatment can lead to significant morbidity or death.
- Cessation of the suspected offending neuroleptic pharmacologic agent.
- Treatment of hyperthermia(Fever) with cooling blankets or ice packs to the axillae and groin may be needed.
- Aggressive hydration is often required, to prevent disseminated intravascular coagulation (DIC) secondary to rhabdomyolysis, deep venous thrombosis especially if highly elevated CPK levels threaten to damage the kidneys.
- All dopamine agonists given to reverse the hypodopaminergic state.

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- Bromocriptine Mesylate, a dopamine agonist,
PO /NG tube starting dose 2.5mg twice /thrice daily Max-45mg.
- Amantadine starting dose -100mg twice/thrice daily Max- 600mg
- Dantrolene a muscle relaxant that works by inhibiting calcium release from the sarcoplasmic reticulum.
starting dose PO 50mg -200mg.
- Benzodiazepines, which can be helpful in controlling agitation but may also ameliorate symptoms and hasten recovery in milder cases.

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What is the treatment, since it is an Neurological emergency delay should not be done and treatment should be given as soon as possible. Cessation of the suspended offending agent that is the first thing that we should be doing it. Treatment of the hyperthermia that is fever by cooling blankets or ice packs, aggressive hydration, and a dopaminergic agonists agent to counteract the hypo dopaminergic state.

Now these are this is basically given by bromocriptine or amantadine. Bromocriptine is given 2.5 milligram twice or thrice daily and amantadine 100 milligram twice or thrice. Dantrolene is a muscle relaxant it works by inhibiting calcium release from sarcoplasmic reticulum it is also given to counteract the condition. It leads to decrease in the muscle contraction on the

part of patient. Benzodiazepines also has a muscle relaxant action. So, these are also given to decrease the agitation of the patient.

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Antipsychotic Overdose

- 'Antipsychotics,' Aka "Neuroleptics" are a broad category of medications initially developed to treat psychosis arising from a large spectrum of psychiatric and organic causes .
- Other indications such as headaches, Tourette syndrome, and post-herpetic neuralgia.
- Neuroleptic medications can be broadly divided into two categories.

Based largely on their pharmacologic properties, and, therefore, their side effect profiles:

First-generation antipsychotics (FGAs), also known as	'Typical' antipsychotics
Second-generation antipsychotics (SGAs), termed	'Atypical antipsychotics

Side effect Profile

Sedation	Due to Histaminergic receptors
Weight gain	Due to Histaminergic receptors, 5HT _{2C} (Atypical)
Erectile dysfunction in male	Decrease in serotonin ,dopamine
Oligomenorrhea or amenorrhea in female	(decreases dopamine thereby increasing prolactin)
Orthostatic hypotension	alpha 1 blocking action
Constipation	due to cholinergic receptors
ECG changes -	(QT prolongation, torsades de pointes-PSVT)

Sedation:	Chlorpromazine> Quetiapine> Olanzapine> Risperidone>Haloperidol
Hypotension:	Clozapine>Risperidone>Quetiapine> Aripiprazole
Q T Prolongation:	Antipsychotics
	Cariprazine , lurasidone --->aripiprazole, clozapine , olanzapine--> amisulpride, haloperidol, quetiapine-->

Antipsychotic overdose, antipsychotic basically typical and typical with those neuroleptic medications they are basically they are into typical antipsychotics and atypical antipsychotics those first generation and second generation. So, depending upon the side effect profile, the receptor mediated action on the drugs we can actually try to observe or assess, okay, which drug can be given to the, which drug might have which drug with the patient might have consumed who is there at the emergency.

So, depending upon those receptor mediated action of the drugs, we can actually observe the patient. So orthostatic hypotension is basically due to alcohol and blocking action. This

erectile dysfunction in male it is due to serotonin decrease in the serotonin and dopamine levels, weight gain is preferentially most commonly seen due to 5HT_{2C} receptors. The hypertension which is observed in the patient is most commonly due to close up in sick whereas Risperidone, Quetiapine, Aripiprazole they are less commonly notorious to cause hypotension, among Q T prolongation most notorious is to cause by the pimozide which is the typical antipsychotic and cariprazine and lurasidone they are very least possible factors very lease possible agents which are causing to deplogation.

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No specific antidote and Supportive treatment is the mainstay of Antipsychotic toxicity.

Consider gastric lavage or induction of emesis as soon as possible and followed by the administration of activated charcoal.

Maintenance of Airway, Breathing, and circulation are critical factors for survival.

A patent airway is ensured by using an oropharyngeal airway or endotracheal tube.

Comatose patients with a difficult airway or upper airway obstruction require a tracheostomy.

Supplemental oxygen is administered via nasal prongs or facemask.

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Hypotension and circulatory collapse need aggressive treatment with intravenous fluids, concentrated albumin, and vasopressor agents such as norepinephrine or phenylephrine.

Extrapyramidal reactions such as acute dystonia are treated with Central anticholinergics.

ECG and vital signs require regular monitoring, especially for signs of torsades de Pointes or QT prolongation, or dysrhythmias.

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So, how will you treat this toxicity since there is no specific antidote, supportive therapy remains the mainstay. We have to consider for gastric lavage or reduction of MSS maintenance of ABC that is airway, breathing and circulation, which is very critical for the

patient, comatose patient with a difficult airway or upper airway obstruction they require tracheostomy and supplemental oxygen if required.

So hypotension and circulatory collapse they need aggressive treatment with IV fluids concentrated albumin and vasopressor agents as norepinephrine or epinephrine. Extrapyramidal reactions they are basically treated by Central Anticholinergics. ECG and vital signs that has to be regularly monitored, especially for signs of polymorphic ventricular tachycardias.

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Antidepressants Related Emergencies

SSRI discontinuation Syndrome

- Abrupt discontinuation of SSRI with shorter half life Paroxetine, fluvoxamine

Dizziness
Weakness
Nausea
Headache
Rebound depression
Anxiety
Insomnia
Poor Concentration
Paraesthesias
Migraine like symptoms

Treatment
Restart the offending agent
Fluoxetine with max half life of 1 week is substituted with the offending agent

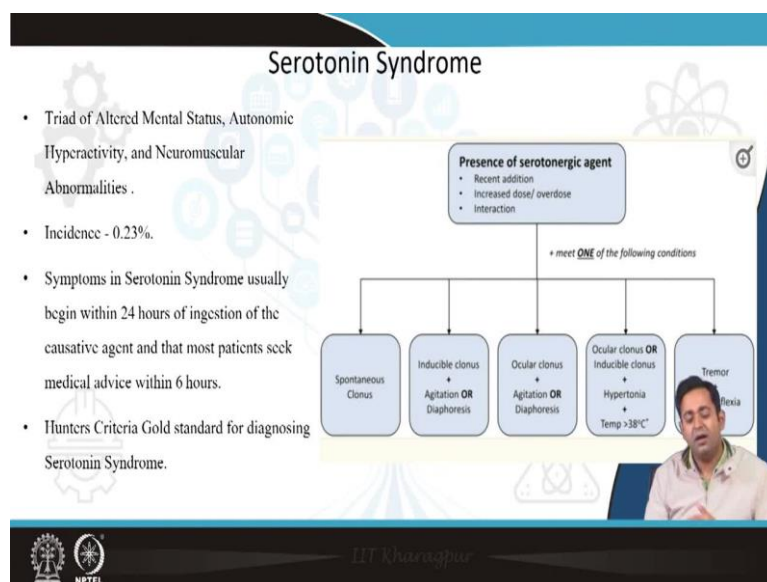
Persons who experience adverse effects in first week of taking SSRI generally experiences discontinuation syndrome

Let us come to antidepressant related emergencies. First and foremost is the SSRI discontinuation syndrome. Now SSRI discontinuation syndrome basically is observed with the ones those who experience symptoms during the first week of starting of the SSRIs.

So these are the patients in which the SSRI discontinuation syndrome is most commonly observed. How it happens if there is abrupt discontinuation of the SSRIs with preferentially shorter half lives of the drugs that is paroxetine or fluoxetine. So, these are basically manifested in the form of weakness, nausea, rebound depression, the symptoms the there is a resurgence of symptoms.

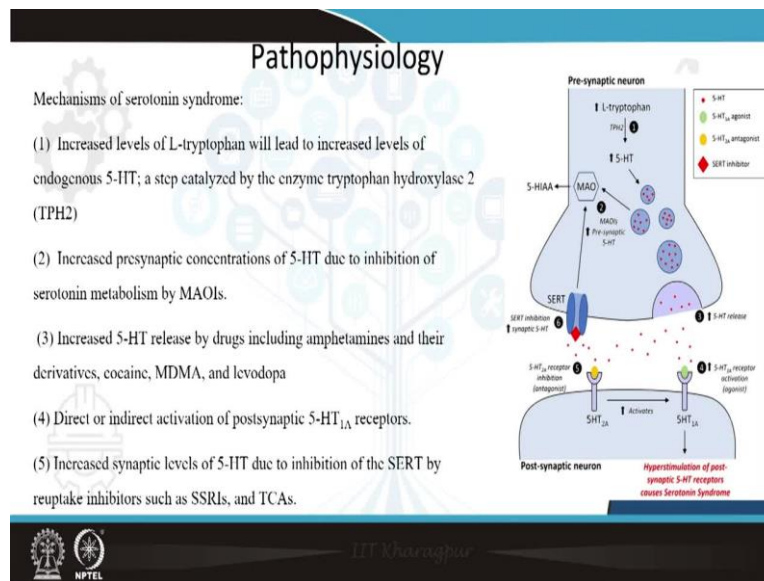
There is insomnia poor concentration agitation, at times can be migraine like symptoms. So they are treated by restarting the offending agent suppose we have abruptly stopped we need to restart it or it is managed with the fluoxetine adding fluoxetine because it has the maximum half life of 7 days more than one week.

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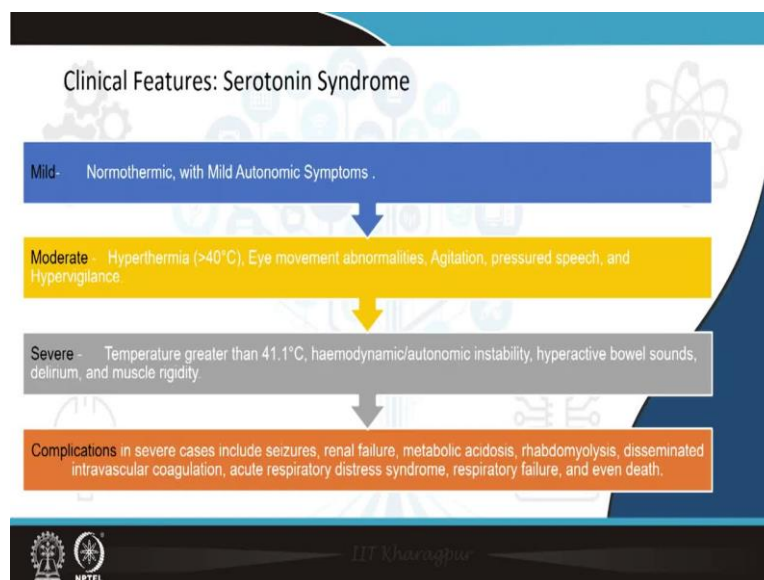
Next come serotonin syndrome, now serotonin syndrome is basically diagnosed with the help of triad that is altered mental status, autonomic dysregulation and neuromuscular abnormalities, there is a hunter's criteria which is utilized for diagnosis of the serotonin syndrome, it is spontaneous clonors agitation diaphoresis with or without hypertonia temperature and tremors and hyperreflexia these all conditions has to be met one or the either cases.

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What is the pathophysiology of serotonin syndrome? The basic idea is that in the synaptic cleft, the serotonin levels are increasing, this can be due to either decrease degradation, or decrease reuptake or do to increase synthesis or various kind of pathological reasons. So, basic idea is the serotonin levels in the synaptic cleft is increased. That is how the serotonin syndrome is caused.

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Treatment

- First step is to identify and stop the offending serotonergic medication.
- Supportive care to stabilize vital signs (maintenance of oxygen saturations, intravenous fluids, and continuous temperature cardiac monitoring).
- Sedation with benzodiazepines (such as diazepam or midazolam/lorazepam).
- In severe cases, consideration of serotonin antagonists alongside muscle paralysis.

In Mild Cases:

In mild cases, discontinuation of the offending medication, sedation with benzodiazepines and supportive care is often sufficient.



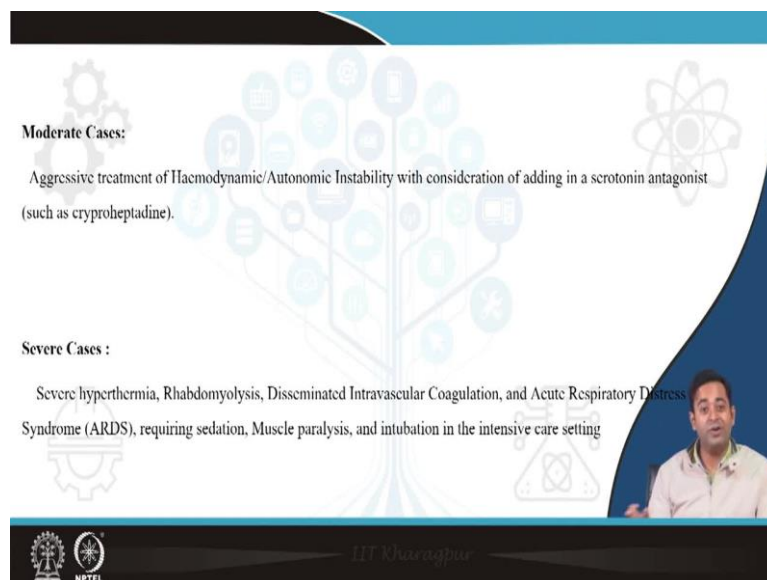
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Moderate Cases:

Aggressive treatment of Haemodynamic/Autonomic Instability with consideration of adding in a serotonin antagonist (such as cyproheptadine).

Severe Cases :

Severe hyperthermia, Rhabdomyolysis, Disseminated Intravascular Coagulation, and Acute Respiratory Distress Syndrome (ARDS), requiring sedation, Muscle paralysis, and intubation in the intensive care setting



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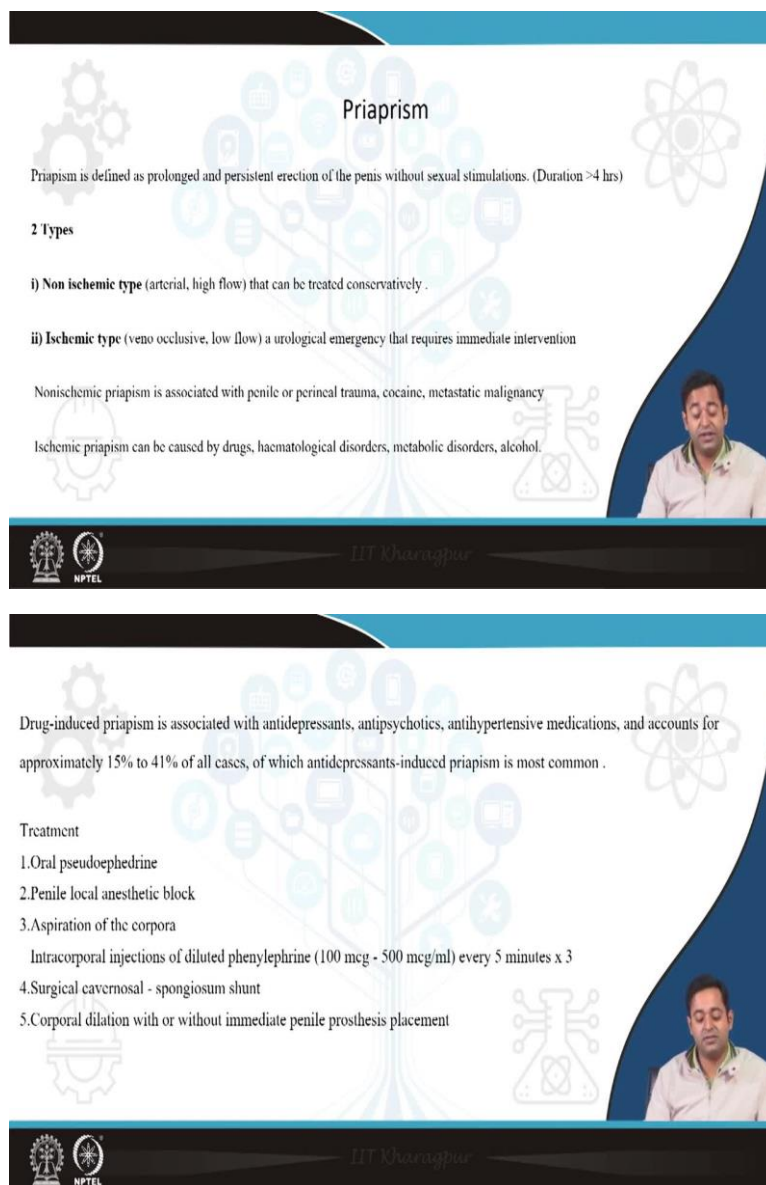
What are the features of serotonin syndrome? They are basically divided into mild moderate and severe types mild is you have mild autonomic symptoms. In moderate subtype you have hyperthermia that is more than 40 degrees Celsius eye movement abnormalities agitation pressured speech, in severe the temperature is more than 41.1 degrees Celsius and then there is autonomic instability with hyperactive bowel sounds at times associated with delirium that is patient is confused and muscle rigidity can also be present. In complicated cases, seizures and human failure with metabolic acidosis or rhabdomyolysis and multi organ failures can lead to death of the patient. How do we treat this?

Treatment is by identifying and stopping the offending serotonergic agent with the patient is receiving supportive care to stabilize vital signs oxygen saturation and maintain his oxygen

saturations. IV fluids, continuous temperature cardiac monitoring, and in severe cases, there is consideration of the addition of certain antagonists alongside the muscle paralysis.

So in mild, moderate severe cases you have in mild cases, you just need to discontinue the offending medication and give supportive treatment. In moderate cases, you have aggressive treatment, you need to aggressively treat the hemodynamic instability, with addition of serotonergic antagonists added Cyproheptadine and in severe cases, since it is a multi organ failure or multiple organ systems are involved. We need to take consultations for the like we need to (()) (39:08) with the other departments in order to intervene the condition on the patient.

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Priapism

Priapism is defined as prolonged and persistent erection of the penis without sexual stimulations. (Duration >4 hrs)

2 Types

- i) **Non ischemic type** (arterial, high flow) that can be treated conservatively .
- ii) **Ischemic type** (veno occlusive, low flow) a urological emergency that requires immediate intervention

Nonischemic priapism is associated with penile or perineal trauma, cocaine, metastatic malignancy

Ischemic priapism can be caused by drugs, haematological disorders, metabolic disorders, alcohol.

Drug-induced priapism is associated with antidepressants, antipsychotics, antihypertensive medications, and accounts for approximately 15% to 41% of all cases, of which antidepressants-induced priapism is most common .

Treatment

- 1.Oral pseudoephedrine
- 2.Penile local anesthetic block
- 3.Aspiration of the corpora
- Intracorporal injections of diluted phenylephrine (100 mcg - 500 mcg/ml) every 5 minutes x 3
- 4.Surgical cavernosal - spongiosum shunt
- 5.Corporal dilation with or without immediate penile prosthesis placement

Other most common, like the condition which is encountered in emergencies or priapism. Priapism is defined as prolonged and persistent erection of the penis without sexual stimulation and this erection is more than 4 hours duration. Now it is basically of 2 types that is non-ischemic and ischemic type. Ischemic type is due to veno occlusive low flow and non ischemic type is due to arterial and that is high flow. Now this non ischemic type is treated conservatively and the ischemic type becomes a neurological emergency.

So since it is a neurological emergency the treatment is by oral pseudo ephedrine penile local anesthetic blockage, aspiration of the corpora that is with the injection diluted phenylephrine is given. Surgical cavernosal is spongiosum shunt is also tried and Corporal dilatation with or without immediate penile prosthesis is placed, if it becomes a condition where the treatment is not possible with other cases.

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Anticholinergic Syndrome

Medications with anticholinergic properties include antidepressants, antiparkinson drugs, antipsychotics,

Common physical exam findings

Flushing, anhidrosis, dry mucous membranes, mydriasis, altered mental status, fever, urinary retention, Decreased bowel movements.

Central nervous system effects may include delirium, hallucinations, agitation, restlessness, confusion, and picking at clothing and bedding.

Seizures and jerking movements are possible

Treatment

1. Physostigmine is usually only given in the case of both peripheral and central signs and symptoms of anticholinergic poisoning.
2. Maintain Hydration.
3. ECG monitoring (TCA's prone to QT prolongation)

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What are the anticholinergic syndrome, but these other medicines which gives rise to symptoms of like those anti cholinergic receptors empty receptors, which is contracting the patient, there are systems which commonly involved are CNS that is the patient can land up in delirium, there can be hallucinations, agitation, restlessness, confusion, picking up the cloth, those kinds of behaviors can be present.

Among physical findings patient might need be like have sweating, or like there will be loss of sweating, dry mucous membranes, altered mental status, immediate attention, these all features are due to Mt receptor blockade, where you have decreased secretions.

So, treatment is by Physostigmine. It is only given in the cases of peripheral and central signs and symptoms of antibiotic poisoning. Second is to maintain hydration and third is ECG monitoring because TCA is notorious to cause Q T prolongation.

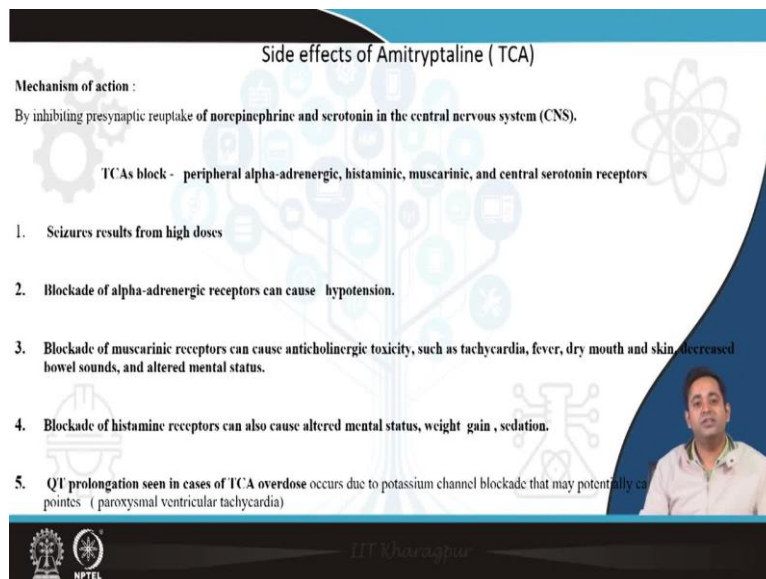
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Antidepressant Overdose

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Side effects of Amitriptyline (TCA)

Mechanism of action :

By inhibiting presynaptic reuptake of norepinephrine and serotonin in the central nervous system (CNS).

TCA's block - peripheral alpha-adrenergic, histaminic, muscarinic, and central serotonin receptors.

1. Seizures results from high doses
2. Blockade of alpha-adrenergic receptors can cause hypotension.
3. Blockade of muscarinic receptors can cause anticholinergic toxicity, such as tachycardia, fever, dry mouth and skin, decreased bowel sounds, and altered mental status.
4. Blockade of histamine receptors can also cause altered mental status, weight gain, sedation.
5. QT prolongation seen in cases of TCA overdose occurs due to potassium channel blockade that may potentially cause pointes (paroxysmal ventricular tachycardia)

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Management

Proper management of airway, breathing, and circulation is critical in cases of TCA poisoning.

Charcoal decontamination may be effective up to 2 hours' post-ingestion, especially if the bowel sounds are diminished.

In cases of Seizures, benzodiazepines are used, but in cases of refractory seizures, prompt administration of anticonvulsants, such as phenobarbital or propofol, or even general anesthesia, should be considered.

Sodium bicarbonate should be given to hemodynamically unstable patients, patients with seizures, and patients with QRS prolongation of more than 100 msec.

The aim of this therapy is to narrow the QRS and to keep the serum pH between 7.5 and 7.55.

Antidepressant overdose and reverse an overdose TCS as we have seen they cause anticholinergic syndrome so we need to be careful regarding the dosage of the TCS when the patient is being given.

So, how are they managed and proper management of the airway, their breathing and circulation in cases of TCA poisoning. In cases of seizures benzodiazepines is used but in case of refractory seizures, the anticonvulsants like phenobarbital or propofol, like GA can also be given sodium bicarbonate should be given to hemodynamically unstable patients and the aim is to narrow down the QRS complex because TCA are known to district cause QT prolongation, QRS complexes prolongations.

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Treatment

- Alpha-adrenergic agents, such as norepinephrine, should be used for cases not responding to hypotension.
- Since TCA are highly protein bound with an extensive volume of distribution, enhanced elimination with dialysis and hemoperfusion is not effective.

SSRI (sertraline)& SNRI's (venlafaxine)

<p style="text-align: center;">Sertraline</p> <p>➤ Mechanism of action : Blockade of Reuptake of serotonin from the synaptic cleft.</p> <ul style="list-style-type: none"> The Hunt serotonin toxicity criteria decision rule describes 5 constellations of symptoms that define serotonin toxicity <ol style="list-style-type: none"> 1. Spontaneous clonus 2. Inducible clonus and agitation or diaphoresis 3. Ocular clonus with agitation or diaphoresis 4. Tremor and hyperreflexia 5. Ocular clonus, hypertonicity, and temperature greater than 38 C 	<p style="text-align: center;">Venlafaxine</p> <ul style="list-style-type: none"> Mechanism of action : Venlafaxine works by increasing serotonin levels, norepinephrine, and dopamine in the brain by blocking transport proteins and stopping their reuptake at the presynaptic terminal. <p>Venlafaxine is essentially a selective serotonin reuptake inhibitor at 75 mg, and with higher doses such as 225 mg/day.</p> <ul style="list-style-type: none"> Symptoms of an overdose of venlafaxine can include: Tachycardia, unusual sleepiness, dilated pupils, seizures, cardiac arrhythmias, hypotension, muscle aches or pains, or dizziness.
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So, treatment is by alpha adrenergic agents that is norepinephrine and since TCA are a highly protein bound with an extensive volume of distribution enhanced elimination, so, we should not try to eliminate the drug out with the dialysis or hemoperfusion that is why it is not effective.

So, Let us come to SSRIs and SNRIs. How are they acting so like for SSRIs there is those hunts written on top lytic criteria and the features are spontaneous clonus inducible clonus, agitation ocular clonus, tremors hyperreflexia and in case of venlafaxine, you have those tachycardia, unusual sleepiness, dilated pupils, seizures, vomiting, cardiac arrhythmias, hypotension, muscle aches or pains or dizziness.

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Management

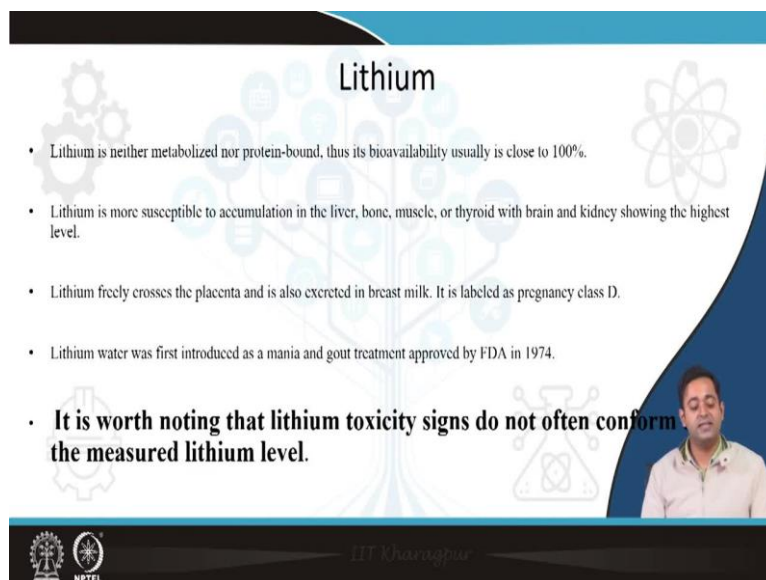
- Patients who were experiencing minor symptoms can be managed at home in conjunction with a doctor or with a regional poison control center.
- Treatment is largely supportive and involves discontinuation of all serotonergic therapies, control of agitation with benzodiazepines and defervescence.
- Hypertonicity, rigidity, and high-grade fever are evident in severe cases of serotonin toxicity where patients require endotracheal intubation and ventilatory support.
- Muscular rigidity is thought to be the primary cause of hyperthermia and death so patients with severe symptoms should be rapidly cooled.
- If benzodiazepines do not sufficiently control muscle spasm or temperature is greater than 41.1 C, intubation with neuromuscular blockade and paralysis is considered.
- In cases refractory to supportive care, cyproheptadine which does not have an IV formulation and must, therefore, be administered via a nasogastric or orogastric tube. (Anti serotonergic agent)

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How can we manage this patients who are experiencing minor symptoms can we manage that home in conjunction with a doctor on a regional poison control center. So treatment is largely supportive and it involves discontinuation of all serotonergic drugs hypertonicity rigidity and hybrid fever are evident in severe cases of certain toxicity whereas the patients requiring (()) (43:22) intubation.

So muscle rigidity is the primary cause for hyperthermia. We need to counteract this, if benzodiazepines is like they do not sufficiently control spasm or temperatures more than 41 degrees Celsius, we need to intubate the patient and in cases of refractory to supportive control, so a supportive care Cyproheptadine is indicated.

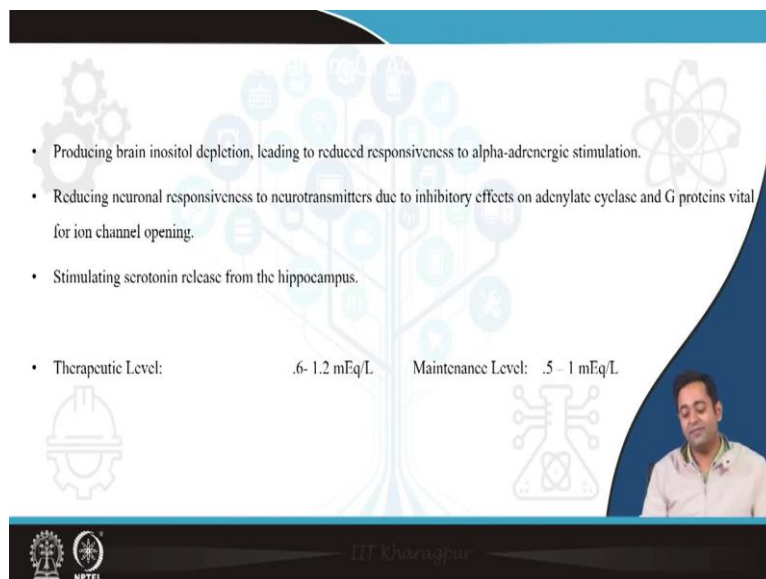
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Lithium

- 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 water 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 the measured lithium 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: .5 – 1 mEq/L
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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

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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 Arrhythmia)

Severe Lithium Intoxication (Lithium Level >2.5 mEq/L)

- Generalised Convulsions
- Oliguria and Renal Failure
- Death

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Among mood stabilizers, lithium and valproate are most commonly given for bipolar affective disorders or for augmentation of the major depressive disorders, so lithium, the most important point is that despite of the fact is lithium serum levels are within the normal range, but the side effects of lithium are many manifested. So the therapeutic level for lithium is 0.6 to 1.2 milligrams per liter and the maintenance level is 0.5 to 1 milligrams per liter.

So what are the signs and symptoms where the patient with the lithium toxicity with mild and moderate intoxication they can present with, they can be in the form of gastrointestinal that is vomiting, abdominal pain, dryness of mouth, or neurologic manifestations like ataxia. Dizziness, slurred speech, misdiagnosis, muscle weakness.

In cases moderate and severe intoxication, gastrointestinal manifestations in the form of anorexia patient will not be able to eat there is no hunger or appetite is persistent nausea,

dizziness, vomiting on the part of patient in neurologic manifestations you have blurred vision, diplopia, hyperactive tendon reflexes, convulsions might develop seizures. Patient can be confused at times and can go to coma. In case of severe lithium intoxication, there can be generalized convulsions, but all cortical lobes areas are involved, patient can end up in acute renal failure or 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 are we going to treat this lithium toxicity, lithium toxicity levels when they are above 2 mili equivalents per liter, they give rise to the manifestations more readily. So since there is no antidote, the primary is the primary we need to look for hydration of the patient. So we need to avoid all diuretics and gastrointestinal distress decontamination. If the patient has severe renal dysfunction or failure, or severe altered mental status, hemodialysis is considered and for tremors 20 to 30 milligrams of propanol 2 to 3 times per day can be given.

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Valproate

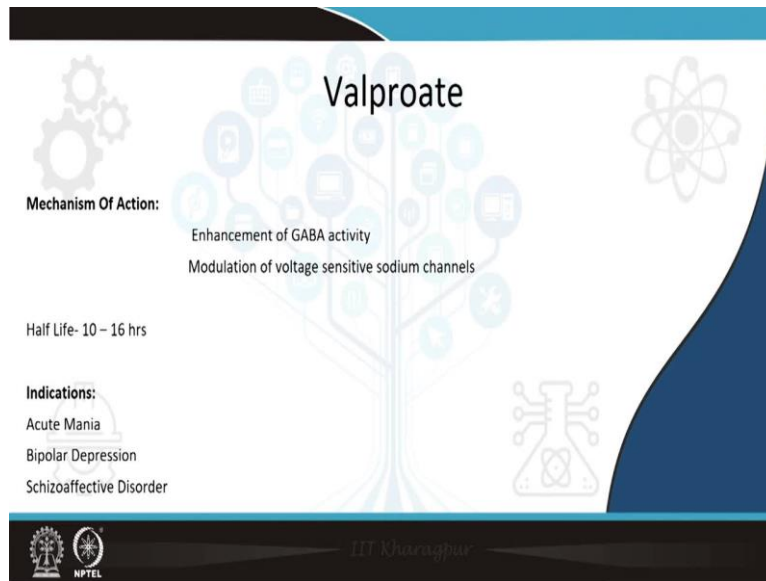
Mechanism Of Action:


- Enhancement of GABA activity
- Modulation of voltage sensitive sodium channels

Half Life- 10 – 16 hrs

Indications:

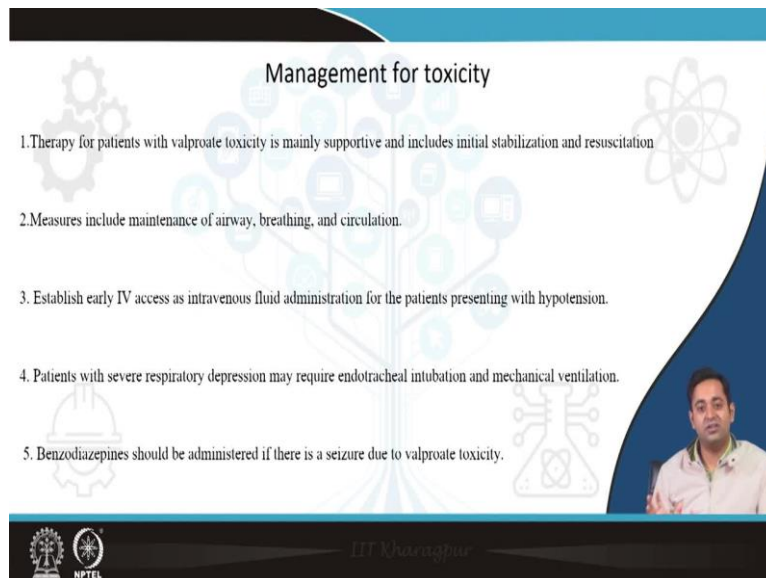
- Acute Mania
- Bipolar Depression
- Schizoaffective Disorder




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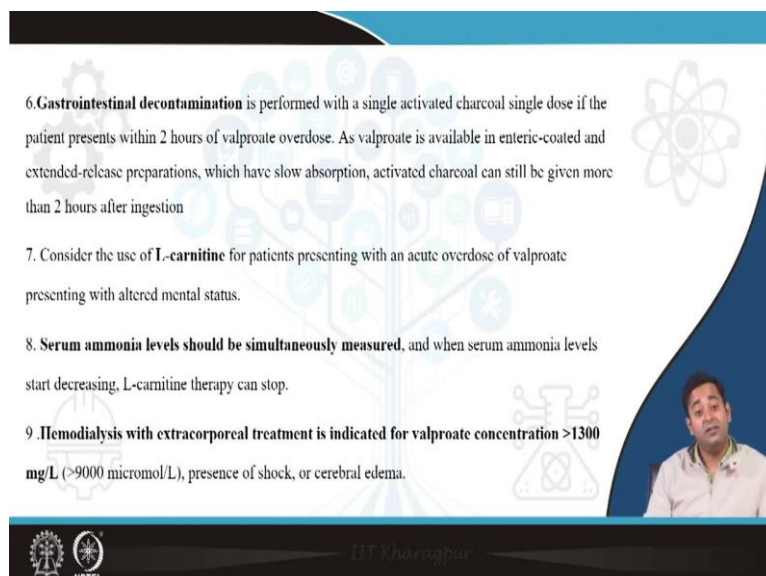
Management for toxicity


1. Therapy for patients with valproate toxicity is mainly supportive and includes initial stabilization and resuscitation
2. Measures include maintenance of airway, breathing, and circulation.
3. Establish early IV access as intravenous fluid administration for the patients presenting with hypotension.
4. Patients with severe respiratory depression may require endotracheal intubation and mechanical ventilation.
5. Benzodiazepines should be administered if there is a seizure due to valproate toxicity.



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6. **Gastrointestinal decontamination** is performed with a single activated charcoal single dose if the patient presents within 2 hours of valproate overdose. As valproate is available in enteric-coated and extended-release preparations, which have slow absorption, activated charcoal can still be given more than 2 hours after ingestion
7. Consider the use of **L-carnitine** for patients presenting with an acute overdose of valproate presenting with altered mental status.
8. **Serum ammonia levels should be simultaneously measured**, and when serum ammonia levels start decreasing, L-carnitine therapy can stop.
9. **Hemodialysis with extracorporeal treatment is indicated for valproate concentration >1300 mg/L (>9000 micromol/L)**, presence of shock, or cerebral edema.



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In case of valproate, therapy for patients with valproate toxicities is mainly supportive and initial stabilization and resuscitation, that is ABC airway manage breathing and circulation, we need to give IV access that is IV administration of patients who are having hypotension. It is IV fluids. Patients with severe respiratory depression may require endotracheal intubation and mechanical ventilation and benzodiazepines are given if there is a chance of seizures due to Belgrade toxicity.

Gastrointestinal decontamination is performed and the use of L carnitine is indicated for if there is an acute overdose of valproate presenting with altered mental status, serum ammonia levels has to be assessed. This can give rise to confusion delirium for the patient, and hemodialysis with the extracorporeal treatment is integrated for valproate concentration and the levels of Alaphard they increase more than 1300 milligrams per liter.

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Benzodiazepines

- Benzodiazepines can have short or long durations of action. This affects the onset and course of withdrawal.
- **Short-acting benzodiazepines** include oxazepam, alprazolam and temazepam. Withdrawal typically begins 1-2 days after the last dose, and continues for 2-4 weeks or longer.
- **Long-acting benzodiazepines** include diazepam, clonazepam and nitrazepam. Withdrawal typically begins 2-7 days after the last dose, and continues for 2-8 weeks or longer.

Symptoms include:

- Anxiety
- Insomnia
- Restlessness
- Agitation and irritability
- Poor concentration and memory
- Muscle tension and aches.

The slide features a blue header, a white background with faint chemical and medical icons, and a video inset of a male speaker in the bottom right corner. Logos for IIT Kharyagpur and NPTEL are at the bottom.

Let us come to benzodiazepines, now benzodiazepines basically they are into short acting, ultra short acting and long acting. For short acting the withdrawal symptoms begin within 1 to 2 days for long acting they begin with between 2 to 7 days. The symptoms which can manifest with withdrawals of this short acting benzodiazepine as they are in the form of anxiety, insomnia, restlessness, agitation, irritability, poor concentration, difficulty in memory and not able to retain properly.

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Signs & Symptoms of Benzodiazepines discontinuation


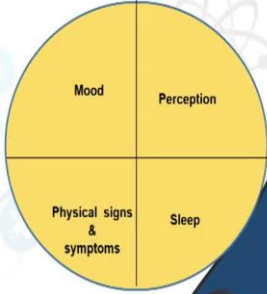
- Return of original anxiety symptoms (Recurrence)
- Worsening of original anxiety symptoms (Rebound)
- Emergence Of New symptoms (Withdrawal)


Disturbances Of Mood and Cognition
Anxiety, Apprehension, dysphoria, pessimism, Irritability

Disturbances of Sleep
Insomnia, altered sleep-wake cycle and daytime drowsiness.

Physical Signs and symptoms
Tachycardia, elevated blood pressure, hyperreflexia, muscle tension, agitation, muscle, joint pain and mal seizures.


Perceptual Disturbances
Hyperacusis, depersonalisation, blurred vision, illusions and hallucinations




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Signs of Benzodiazepines Intoxication


- Inco-ordinaton
- Dysarthria
- Nystagmus
- Impaired memory
- Gait disturbances.
- In severe cases stupor coma and death occurs




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Guidelines for Benzodiazepines Withdrawal

- Evaluate and treat medical and psychiatric condition
- Obtain drug history and urine and blood samples.
- To determine the dose required for stabilisation from history obtained, clinical presentation.
[Most symptoms occur on first 3 days of abstinence and seizure on 2nd or 3rd day]
- Hospitalise if there is poor social support, polysubstance dependence and /or medical problems.
- Long acting benzodiazepines preferred.
- After stabilizing reduce dosage by 30 % on second and third day
- Tapering of the dose should be done depending upon the symptoms present by counteracting them
- Psychological intervention assist patients in managing related anxiety.



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So what are the signs and symptoms of benzodiazepine discontinuation? Basically, we need to look after 4 domains that is mood perceptual abnormalities, physical signs and symptoms and sleep so there can be irregularity in the sleep, the disturbances of sleep. They can be disturbances of mood, and cognition, where you have anxiety, apprehension, difficulty, irritability, then you have perceptual disturbances in the form of hallucinations, illusions, blurred visions, depersonalization.

So what are the symptoms of benzodiazepine intoxication when the patient is coming up with an intoxicated state of taking this benzodiazepines in a high dose, like they can manifest in the form of incoordination their voluntary movement They are not in a position to coordinate hand eye coordination muscle, lower extremities, upper extremities, they are not able to coordinate properly, there is disarticulated, defective articulation of the speech, they are not able to speak properly nystagmus there is to and fro motion of the eyeballs of the patient. Impaired memory, they are not able to like the attention concentration, the registration of the facts and figures, it is not proper, the retrieval of the facts is not proper.

Because these mental faculties they are impaired they are under the effect of this benzodiazepines. There can be difficulty in movement, the gait disturbances, how they work. And in severe cases, stupor, coma, or at times death can also occur.

What are the guidelines for benzodiazepine withdrawal? We need to evaluate and treat the medical and psychiatric condition obtain the drug history with urine and blood samples, determine the dose for which the patient has consumed the particular medicine and for how many days he has, like what is the last time that the dose was consumed, hospitalized if we need to see and collaborative this patient needs inpatient admission for intervention for treatment for the patient is long acting benzodiazepines required or not, and if the patient is taking short acting benzodiazepine, the treatment starts with giving the long acting aspirins because there is no rebound withdrawal for the symptoms.

The patient when they are in the recovery stage, and when these benzodiazepines long acting method regiments are given we need to taper down the dosages with a decrease in the dose of 30 percent on the second and third day, tapering of the dosage should be done depending upon the symptoms present by counteracting them. And at times this psychological intervention assists patient in managing the anxiety.

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Factors which demand psychiatric emergency care

- ☐ Decreased Accessibility
- ☐ Unpredictability
- ☐ Lack of health insurance
- ☐ Poverty
- ☐ Limited education
- ☐ Belongingness from vulnerable population
- ☐ No devotion of separate space for Psychiatric emergencies services in ER
- ☐ Psychiatric patients regarded as nuisance and hindrance to work at Hospital
- ☐ Agitated patients require seclusion and restraints.

Factors related to family mindset and assumptions:

- ☐ **Negative mindset** of the family members to be handled in a separate quiet place
- ☐ **Negative attitude** towards the psychiatric consultant.
- ☐ Assumptions that these symptoms are **not genuine** and are **volitional** which is **not true medical emergency**

Marked change in a person's mental status that activates a response in the support system to seek help and the responsibility of the emergency psychiatry system.

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So what are the factors which demand psychiatric emergency care, these are decreased accessibility unwritten list of the conditions, lack of health insurance, poverty, limited education. We do not know regarding the conditions of the various psychiatric illnesses or the psychotropic drugs.

Belongingness of the vulnerable from the vulnerable population, no diversion of separate space for psychiatric emergency services in the casualty, you do not we do not have separate space to deal with this kind of patient, we have to go and intervene this patient in the common area. Psychiatric patients regarded as a nuisance and hindrance to work at the hospital, they are not given the normal comfort or conducive environment to get treatment.

So negative mindset of the family members at times negative attitude towards the psychiatric consultant and assumptions that these symptoms are not genuine and are volitional on the part of patient they are doing it deliberately, which is not true medical emergency. So Marked change in a person's mental status that activates a response in the support system to seek help may come under the responsibility of the emergency psychiatric system.

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CONCLUSIONS

-In this lecture we have discussed regarding the concepts of suicide , violence/aggression, catatonia , withdrawal /intoxication due to substances, serotonin syndrome, neurolept malignant syndrome , lithium toxicity.

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So in this lecture, we have discussed regarding the concepts of suicide, violence, aggression, catatonia, various deleterious side effects of psychotropics, and its related emergencies these are the references. Thank you