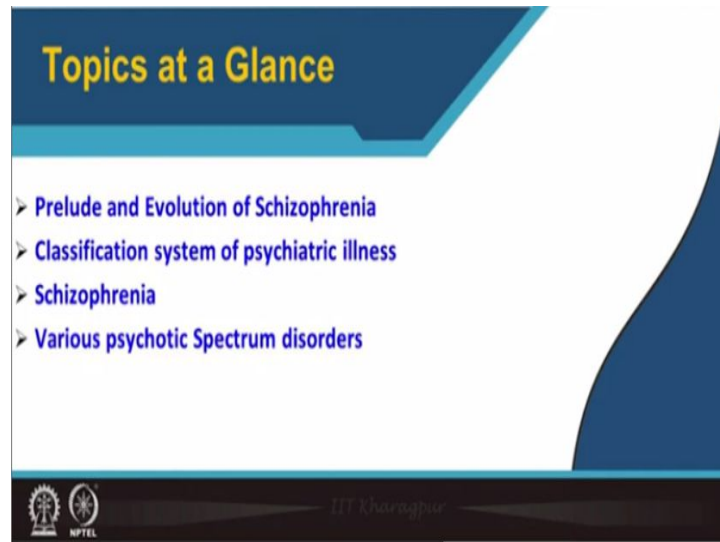


Basics of Mental Health and Clinical Psychiatry
Professor Dr. Sumit Kumar
Department of Psychiatry
Tata Main Hospital, Jamshedpur
Lecture 14
Schizophrenia

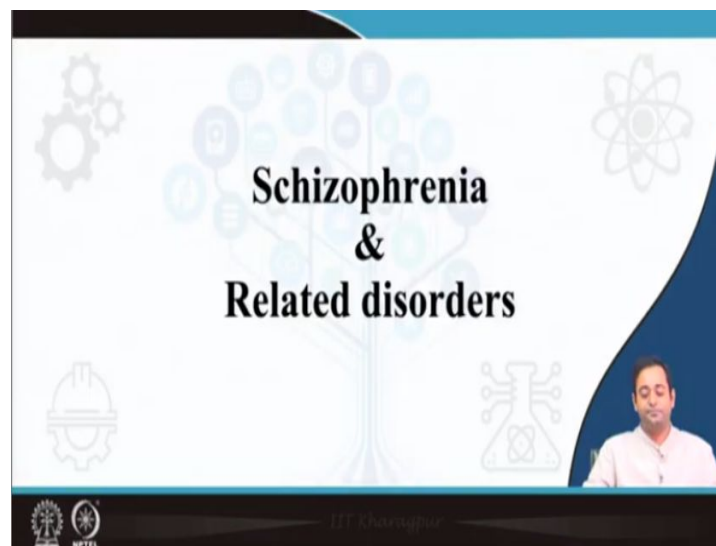
Hello everyone. Let us start lecture number 14. The topic is schizophrenia.

(Refer Slide Time: 0:31)



Let us look at the topic that we will be discussing. Prelude and evolution of schizophrenia. Classification system of psychiatric illness, schizophrenia, various psychotic spectrum disorders.

(Refer Slide Time: 0:44)



Prelude

- Schizophrenia is a disorder with variable phenotypic expression and poorly understood, complex etiology, involving a major genetic contribution, as well as environmental factors interacting with the genetic susceptibility.

presentation 1/1/2020

What is schizophrenia? Schizophrenia, as we all know is a disorder with variable phenotypic expression and poorly understood complex etiology. It has multi multifactorial etiology, involving a major genetic contribution as well as environmental factors interacting with the genetic susceptibility.

(Refer Slide Time: 1:05)

Evolution of Schizophrenia

Emil Kraepelin German psychiatrist (late 19th and the early 20th century)

Gave the term "Dementia Praecox".

He Proposed that all clinical cases have common patterns which results in severe cognitive and behavioural decline and elaborated 9 different forms.

Considered abandoning categorical disease model and replacing them with dimension based model.

Eugene Bleuler Swiss Psychiatrist modified Kraepelin's concept to include

Non affective psychoses & Atypical depressive or manic episode.

Coined term "Schizophrenia"

Proposed **Basic** (obligatory - 4 A's) & **Accessory** (supplementary - hallucinations and delusion) symptoms

4 A's - Autism, Association, Ambivalence, Affect

German Psychiatrist Kurt Schneider, who proposed First Rank Symptoms (FRS) of Schizophrenia in 1959

Audible thoughts, voices commenting on actions, voices arguing, made impulse, made will, made volition, delusional percept, somatopassivity

presentation 1/1/2020

Now, there are various psychiatrists which have actively contributed in this evolution of schizophrenia. Let us try to look at them. Now we will, we will first start with Emil Krypton. Now, he was a German psychiatrist who in, in the late nineteenth and twentieth century gave the term Dementia Praecox. And this Dementia Praecox actually came from Dementia Praecox which, was actually given by Benedict Morel. He was a French psychiatrist.

So, this Emil Krypton proposed that all clinical cases have common patterns which results in severe cognitive and behavioural decline, and they elaborated into nine different clinical subtypes. He was the first person who actually, considered abandoning the categorical subtypes and replacing them with the dimensional disease model.

So, categorical subtypes means they are ways, various subtypes, which has become obsolete right now that catatonic schizophrenia, simple schizophrenia, hyper phrenic schizophrenia, paranoid schizophrenia. So, he actually wanted to replace them with the dimensional model. Dimensional model actually has the illness which is on a continuum basis. There is no separate distinction between the, this, severe subtypes of schizophrenia.

So, what was Bleuler telling? He was a (psych) Swiss psychiatrist. He actually modified the concept of Kraepelin to include non-effective psychosis and a typical depressive or manic episodes. Now we will talk about this bipolar concept, manic and depressive episodes later on. He gave the concept of the, he called the term schizophrenia and, propose the basic and accessory symptoms.

Now, as we better known as the basic symptoms, as we are better known as four a's, which is very commonly known, four a, the first a is autism, association second, third is ambivalence and the last is affect. Now the third person was Scott Schneider. He was a German psychiatrist. He gave the pro, like he proposed the first rank symptoms of schizophrenia.

The first rank symptoms were audible thoughts, voices commenting on actions, voices arguing, made impulse, made will, made, volition, delusional percept and somatic passivity. Let us talk about this in very brief. Somatic passivity is when the patient is actually experiencing thisma feeling that somebody is controlling my body.

So, your body is so here and you have been passively controlled, active, and passive. So, active is, when you are actively doing it, I am holding this pen. I am actively holding this, this pen, and passively means somebody else is doing it. There is an external, a agency or force, which is actually behind this, lifting the pen. So, I am being passively controlled. So, there is somatic passivity.

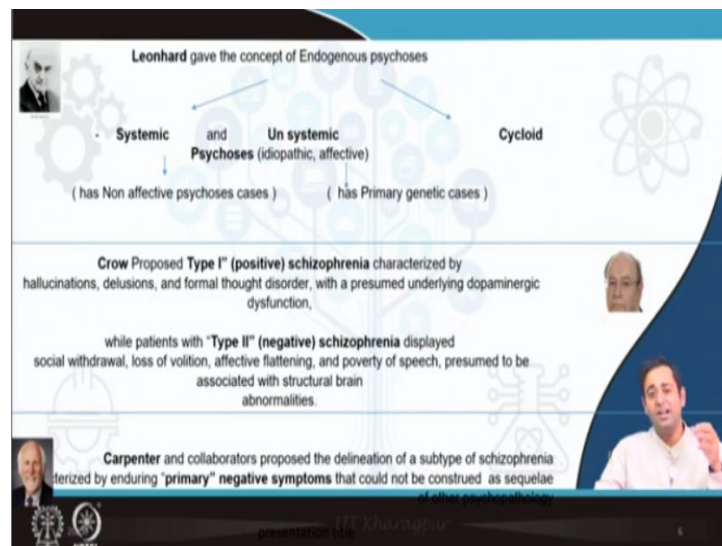
What is delusional percept. Delusion percept is, there is two membernance. Two things which is happening with the delusion percept. First is you have a perception, a normal perception, and then you have a delusional misinterpretation of the normal perception. So, there, there might be something that, I might be having a normal perception of this, driving in a (tra), I

am driving my car and I am on onto some traffic signal. So, there is a red light in the traffic signal. So, suddenly I see that red light. I see, this red light is because of something which is going to have effect on me. Somebody is going to come and kill me. Now suddenly this light is red switched on, so I will be getting persecuted or I will be getting harmed, or I will be getting killed. So, they will have self referential ideation. That is dilution percept, two memberedness.

Made will, made volition, made impulse. So, this is a made phenomena where you have impulse, this impulse are not me because of me, it is the external agency, external force which is actually making this impulse. Made volition means what if I am trying to get up from here, if I am going, if I am trying to walk. So, this evolution is not because of me, I am not doing it. There is some external agency, external force, which is actually making me to do this.

Likewise, you have audit audible thoughts. Audible thought means eco. I was, referring, that, if you have first person auditor hallucination, that means your thoughts are being echoed. These are audible thoughts and voices commenting on action means you have second person auditor hallucination. There is somebody and he is talking, he is commenting on your actions. Suppose you are lifting this pen. So, there will be voices in my ear that see this person is lifting his pen. So, I am being referred as a second person and the person who is being commenting on my action is the first person.

(Refer Slide Time: 5:54)



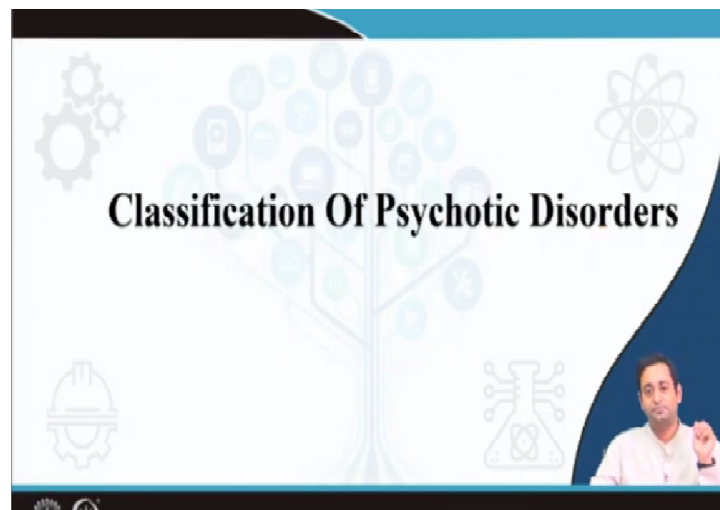
There was just another person Leonhard. Leonhard gave the concept of endogenous psychosis. So, he divided this endogenous psychosis into systemic, unsystemic and cycloid

psychosis. Now, systemic was actually having the non-effective psychosis cases and unsystemic had the primary genetic cases. Crow proposed positive and negative schizophrenia.

Positive schizophrenia means you have all those positive kind of feelings, means delusions, hallucinations, formal thought disorder. So, and the second is negative. That is type two. Type two is when the patient is having negative symptoms. When you are actually not involved, you are not actually conversating with your friends, your families, you have a self-absorbed nature.

You are not having good quality speech. The content of speech is very, very low. So, these are negative symptoms. Third is what the Carpenter gave the concept of primarily negative symptoms. So, there is no positive symptoms at all and there is predominant of negative symptoms.

(Refer Slide Time: 6:53)





Let us look at the classification of psychotic disorders. Now this classification is basically done by this classification system is given by two, two bodies. One is ICD, that is International Classification of Diseases, and the third is, second is Diagnostic and Statistical Manual of Mental Disorders.

So, DSM was given by American Psychiatric Association and ICD was given by WHO world Health Organization. ICD is basically for India and European countries, and DSM is in American continent. There are 22 chapters in ICD and the F code in ICD belongs to the mental and behavioural disorders.

(Refer Slide Time: 7:30)



Current conceptualization of Psychosis:

Refers to the presence of delusions, hallucinations without insight, or both.

- According to current diagnostic classification systems

Impaired reality testing remains central conceptually to psychosis.

Psychosis can be considered to be a set of symptoms in which a person's mental capacity, affective response, and capacity to recognize reality, communicate, and relate to others is impaired.

10

Now, what is psychosis? We are actually talking about what is the basic reason for having, how can we tell that, this patient is psychotic. Now there has to be an impaired reality testing. Impaired reality testing remains a central conceptual to psychosis, and this is actually being proposed by both the bodies, ICD and DSM. They actually give this impaired reality testing as a predominant reason to tell that this person might have a psychotic illness.

Now, what is a psychosis? It can be considered to be a set of symptoms in which a person's mental capacity, effective response and capacity to relate and recognize the reality communicate with others is actually impaired.

(Refer Slide Time: 8:18)

Diagnostic and Statistical Manual Of Mental disorders (DSM) V

Schizophrenia Spectrum differ

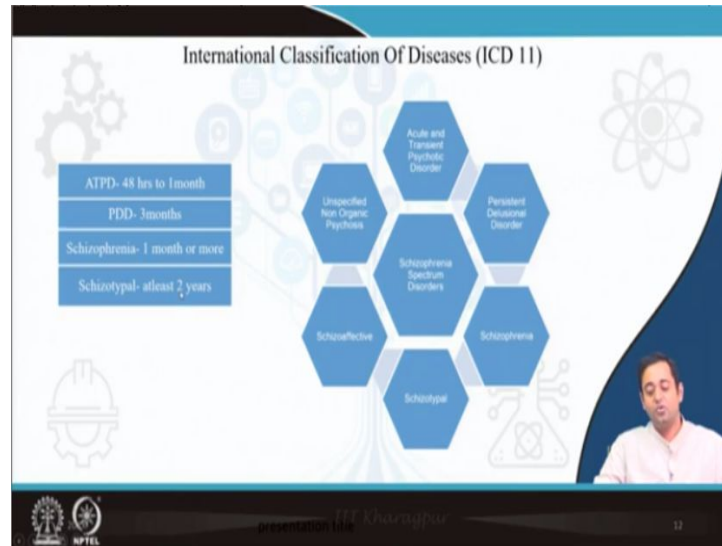
Type	Number	Complexity	Severity	Duration	Features
Brief Psychotic Disorder	1	Low	Mild	At least 1 day to 1 month	Delusions, hallucinations
Unspecified Schizophrenia	2	Low	Mild	At least 1 month	Delusions, hallucinations
Delusional disorder	3	Low	Mild	At least 1 month	Delusions
Schizophrenia Spectrum Disorders	4	Low	Mild	At least 1 month but less than 6 months	Delusions, hallucinations
Schizophrenia	5	Low	Mild	At least 6 months	Delusions, hallucinations
Schizophreniform disorder	6	Low	Mild	At least 1 month but less than 6 months	Delusions, hallucinations
Schizoaffective Disorder	7	Low	Mild	At least 1 month but less than 6 months	Delusions, hallucinations, mood symptoms

11

There are various, different types of ice, psychiatric disorders and the both the classificatory systems they have their own way of describing them. So, in DSM, these all psychotic

spectrum disorders are, they are deferred in terms of type, their number, their complexity, severity, duration, and feature. Brief cycle disorders is the duration is one day to one month. Delusional disorders, at least one month. Schizophrenia form is 1 to 6 month and in schizophrenia it is at least 6 months.

(Refer Slide Time: 8:59)



So, there is a slight difference in ICD, the ATPD what we tell in ICD is actually acute transient psychotic disorders. It is same as root psychotic disorder in DSM. So, there you have 1 day to 1 month, but here in ICD you have 1 48 hours to 1 month. P persistent delusion disorder is 3 months. In DSM you have at least 1 month. So, the duration criteria is different for both the bodies. There in DSM you have 6 months schizophrenia. But here in in ICD you have 1 thought more than 1. Schizotypal is at least 2 years. So, in ICD schizotypal is recognized as a personality disorder.

(Refer Slide Time: 9:43)

Diagnosis: Schizophrenia

ICD

At least one of

1. Thought echo, thought insertion/withdrawal/broadcast
2. Passivity, delusional perception
3. Third person auditory hallucination, running commentary
4. Persistent bizarre delusions

OR two or more of:

1. Persistent hallucinations
2. Thought disorder
3. Catatonic behaviour
4. Negative symptoms
5. Significant behaviour change

Duration: More than 1 month

DSM

At least one of

1. Bizarre delusions
2. Third person auditory hallucinations
3. Running commentary

OR two or more of:

1. Delusions
2. Hallucinations
3. Disorganized speech
4. Grossly disorganized behaviour
5. Negative symptoms

Duration: 1 month of characteristic symptoms
With 6 months of social/occupational dysfunction

Now there are various diagnostic criteria given by both the bodies ICD and DSM.

(Refer Slide Time: 9:51)

ETIOLOGY

I. Genetic: 1st degree relatives high genetic loading risk decreases with 2nd and 3rd degree relatives

II. Social and Environmental:

- i) Social Class
- ii) Deprivation
- iii) Urbanisation
- iv) Childhood adversity (Child sexual abuse – hallucinations, Bullying – Persecutory Delusions)
- v) Migration and ethnicity (Synergism between urbanicity and familial liability)
- vi) Life Events

3 weeks prior to the onset of acute symptoms
experiencing life events doubles the risk of developing schizophrenia over 6 months.

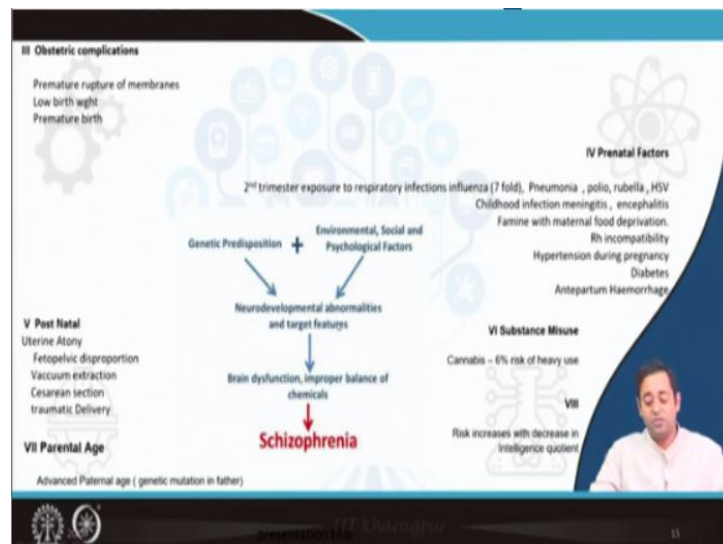
Let us come to etiology. Now there are multifactorial reasons. There are multifactorial reasons for the development of schizophrenia. It is not that a single gene is responsible for schizophrenia. There are pool of genes and this pool of genes actually is responsible in a collective fashion. So, it is not just one gene, which we actually, have this notion, this is the gene which is responsive for skills schizophrenia.

So, till now the studies have been done and they are of a proposition that there is no single gene which is causing (skill) schizophrenia. What are the social and environmental factors? Social class, deprivation, urbanization, childhood adversity. In childhood adversity, the child

sexual abuse in early ages. If child is getting undergoing sexual abuse later on when the schizophrenia develops, there can be more of hallucinations rather than delusions. And if the child is getting bullied, there are evidences, there are literature available which tells that if the child is getting bullied early on, there can be development of persecuted delusions rather than hallucinations.

There is a contribution of migration and ethnicity also. Next year's life events three weeks prior to the onset of acute symptoms. Now this three weeks prior to the onset of acute symptoms holds a very important significance in experiencing life events which doubles the risk of risk schizophrenia over 6 months.

(Refer Slide Time: 11:21)



There are various other reasons, obstetric reasons, while the child is getting born there is they can be premature rupture of membranes, low birth weight, premature birth, postnatal causes various obstetric reasons. Feto, fetal pelvic proportion, dis disproportion, traumatic delivery, cesarean sections. All these contribute to development of schizophrenia. Parental age is a very important thing. Advanced paternal age, which has a genetic mutation which, which has this important contribution in development of schizophrenia.

Next you have prenatal factors. In prenatal factors, it is the respiratory infection, specifically influenza infection which holds sevenfold more times the other respiratory infection which causes schizophrenia. Pneumonia, polio, rubella HSB as we all are contributory factors. RH incompatibility, hypertension during pre pregnancy, diabetes, antepartum haemorrhage. These all contribute.

Substance misuse among all the substances. Smoking that is tobacco and cannabis, they add to the development of schizophrenia. And lastly, risk increases with the decrease in the intelligent quotient. So, as you see, there is genetic predisposition, environmental, social and psychological factors. They act in synchrony to develop schizophrenia. And how are this developing? It is a neurodevelopmental abnormalities and target features which actively contributes to brain dysfunction, improper balance of chemical.

(Refer Slide Time: 12:55)



Now what are the signs and symptoms of schizophrenia? Positive symptoms. There are negative symptoms and there are cognitive symptoms. In positive symptoms we all know delusion is there, hallucinations, conceptual disorganization, patient is not able to think properly. There are, there is problem in the analyzing ability. There is catatonia. Catatonia is a very important neuro, neuro psychological condition which, happens or happen in schizophrenia as well as in biological this bipolar disorders.

So, in catatonia, patient might experience mutism negativism. There is equal aquip apraxia, there is posturing, all those kinds of things. There is severe suspiciousness and persecution which the patient might feel that he or she might be persecuted or there are persons after him. When you are talking about negative symptoms, there there can be a (hell), there can be a lot of host of reasons where patient might experience this negative symptoms in the form of blunted effect.

They are very dull. They are very, very sulky. They do not interact with their family members, their family, their friends at school and college or when you are at at your workplace there is no rapport with your fellow mates, your families and there is difficulty in

What are the cognitive functions? Cognitive function means you are not able to do your normal calculations, your processing speed, your learning ability, your memory. These all get affected very sequentially.

EPIDEMIOLOGY

Onset earlier in Men 10 -25 years

Females much later 25-35 years.

Prevalence 1 % in USA

4/1000 in UK

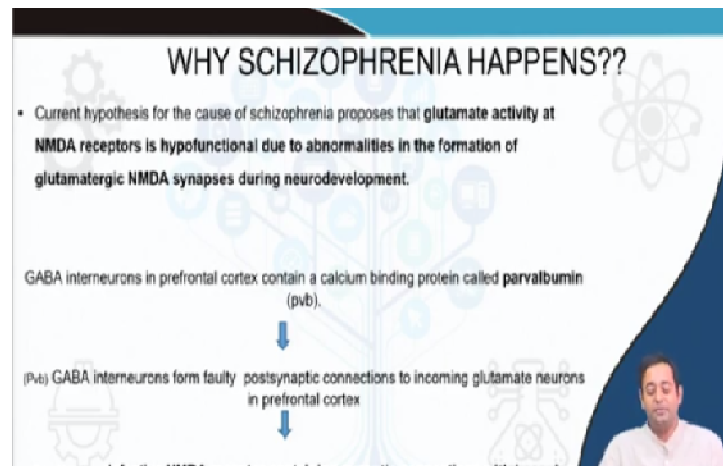
1.5-2.5/1000 in India



Dr. Khanna

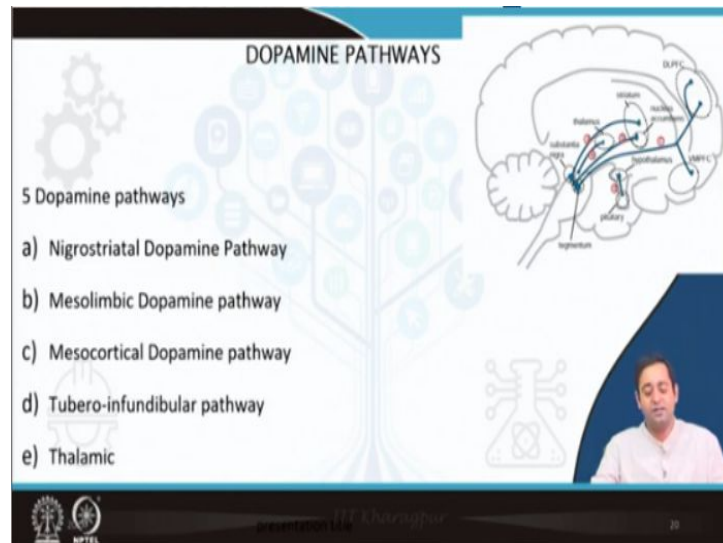
18

(Refer Slide Time: 14:50)



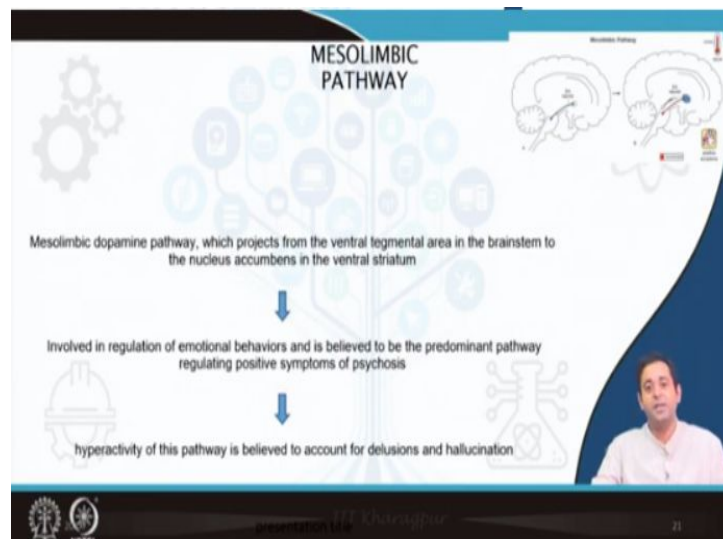
Now why is schizophrenia happens? Current hypothesis is because of the glutamate activity that is NMDA receptors is hyper functional due to abnormalities in the formation of glutamatergic NMDA in its synapses During neurodevelopment. This GABA interneurons in prefrontal cortex contains a calcium binding protein called parvalbumin. Now this parvalbumin GABA interneurons form faulty connections to incoming glutamate neurons in prefrontal cortex. This results into defective NMDA receptor synaptic connections with the incoming pyramidal neurons.

(Refer Slide Time: 15:25)



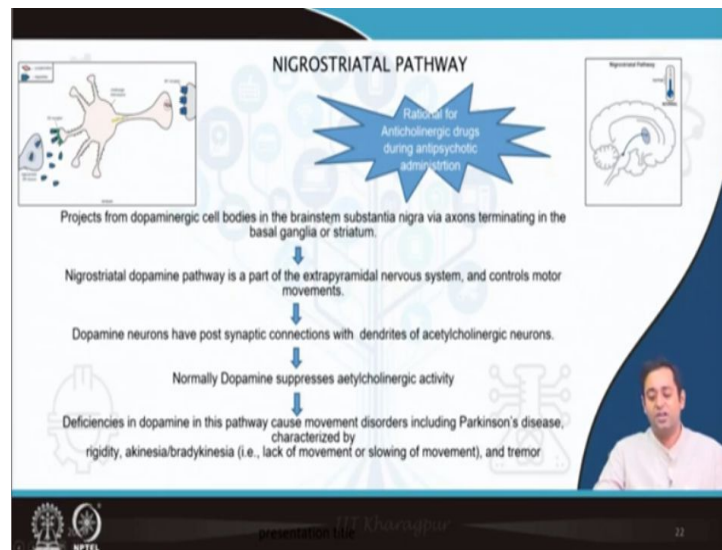
There are five dopamine pathways in the brain; nigrostriatal, mesolimbic, mesocortical, tubero-infundibular and thalamic.

(Refer Slide Time: 15:34)



Mesolimbic pathway is basically which projects from ventral tegmental area. This is ventral tegmental area to the brain nucleus accumbens in the ventral striatum. And this is involved in emotional behaviours which is believed to be the predominant reason for the positive symptoms of psychosis, which actually leads to hyperactivity and is accounting for delusion and hallucinations.

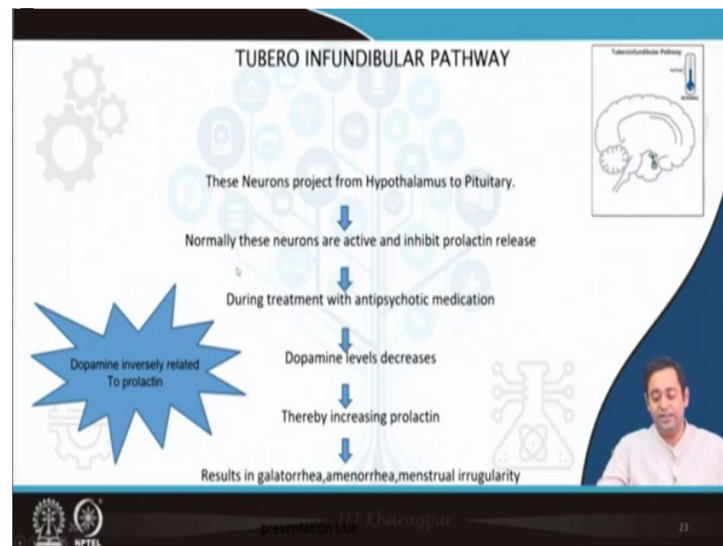
(Refer Slide Time: 16:01)



Nigrostriatal pathway. This projects from dopaminergic cell bodies in the brainstem sub substantia nigra via axons terminating basal ganglia or stratum. These, this nigrostriatal pathway is actually the extrapyramidal nervous system which controls the movement. So, this dopamine neurons, they have a postsynaptic connections with dendritic acetylcholinergic neurons. So, they are under feedback negative inhibition. So, dopamine is more, the dopamine is actually trying to control the acetylcholi.

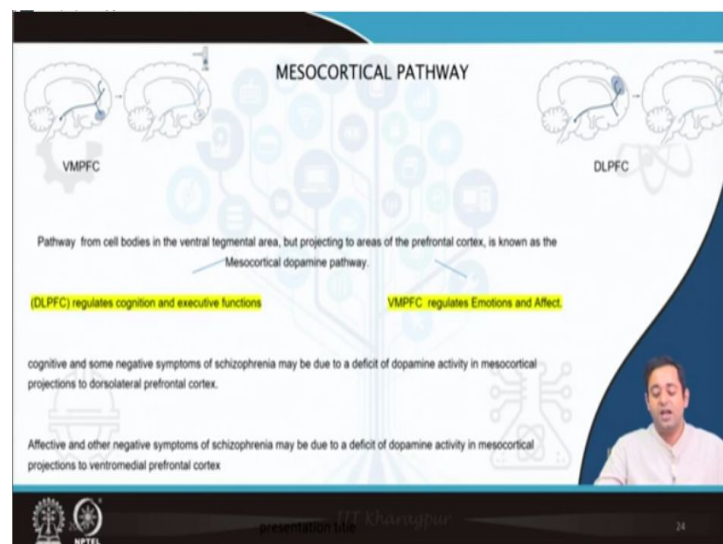
So, what is happening when we actually try to treat patients, this psychotic patients, we actually give antipsychotics. So, antipsychotic they decrease the level of do dopamine and eventually when this dopamine is decrease, so there is release in there is feedback release of this acetylcholinergic levels which actually results into extrapyramidal symptoms in the form of tremors and (reg) rigidity. That is why the rationale for anticholinergic drugs during antipsychotic administration is this pathway which actually contributes to the movement disorders.

(Refer Slide Time: 17:07)



What is tubero-infundibular pathway? These neurons project from hypothalamus to pituitary. These neurons are active and they are actively inhibiting the prolactin release normally and during treatment with antipsychotic medication, dopamine level decreases, thereby increasing the prolactin levels. So, normally this dopamine is inversely related to prolactin. So, when antipsychotic medications are given, they actually decrease the dopamine level. So, when the dopamine level is decrease, prolactin is inversely increased, which later results into galactorrhea, amenorrhea and menstrual irregularities.

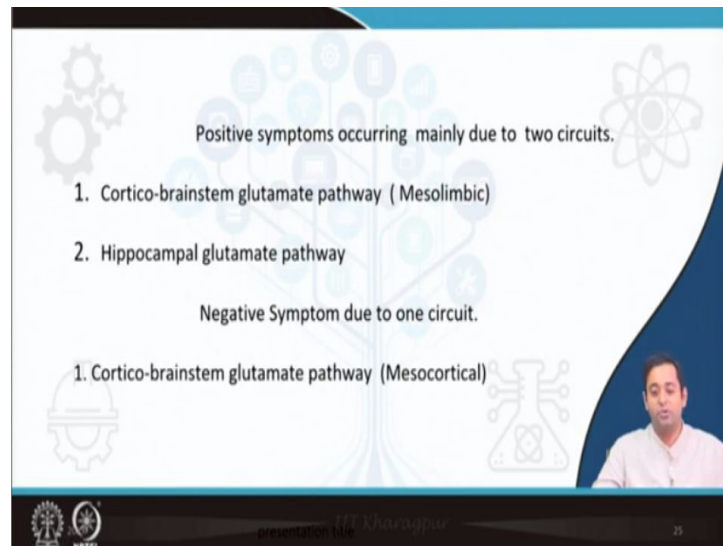
(Refer Slide Time: 17:40)



Let us talk about mesocortical pathways. Mesocortical pathway basically governs ventromedial pathway and dorsolateral prefrontal cortex and ventable prefrontal cortex and

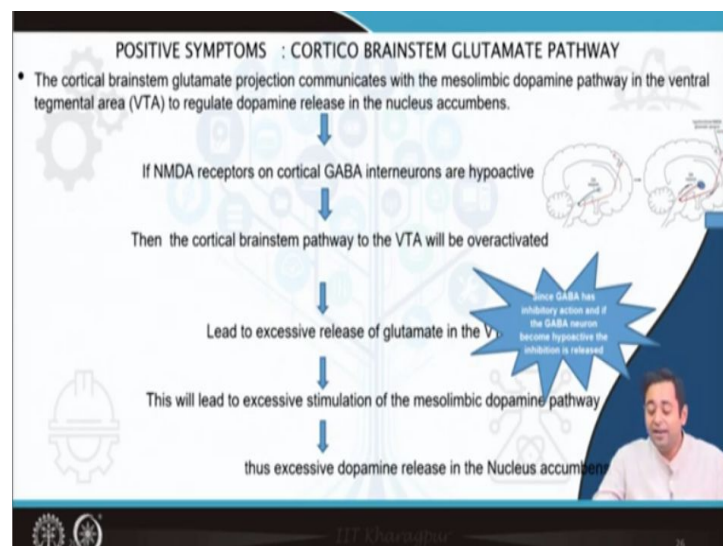
dorolateral prefrontal cortex is basically responsible for cognitive and executive functions. And ventromedial pathway is basically responsible for emotional and effective functions.

(Refer Slide Time: 18:00)



Now why is this happening? Why positive symptoms and negative symptoms? Positive symptoms is basically due to two circuits; from cortex to brainstem glutamate pathway and the second is hippocampal glutamate pathway. And the negative reason is due to cortical brainstem glutamate pathway.

(Refer Slide Time: 18:17)

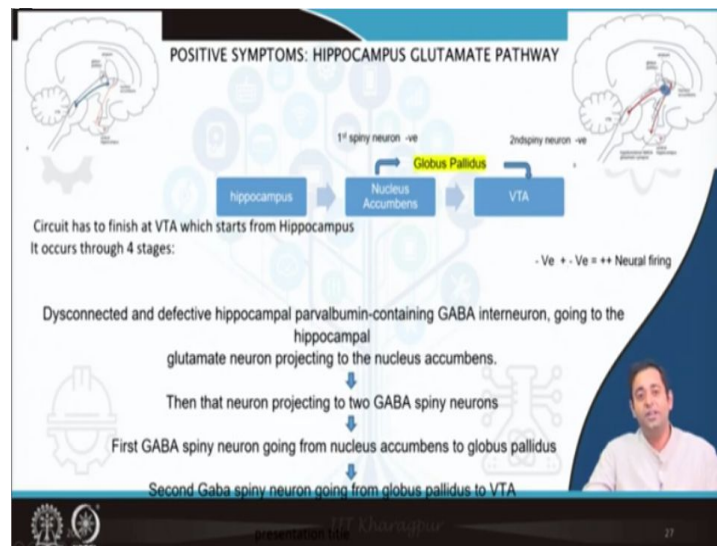


Now what is happening when it is contributing to positive symptoms? Most important reason is cortical, cortico brainstem glutamate projections communicates with the mesolimbic dopamine projections in the ventral tegmental area to regulate dopamine release in the

nucleus accumbens. If NMDA receptors on cortical neuro neurons are hypoactive, then the cortical brainstem pathways of VTA will be overactive, which leads to excessive release of glutamate and excessive stimulation of mesolimbic dopamine pathway, which causes excessive dopamine release in the nucleus accumbens.

Now since GABA has inhibitory reaction and if this GABA neuron becomes hypoactive, the inhibition which was there in the cortical area is released and that is why it leads to increase in the dopamine in the VTA.

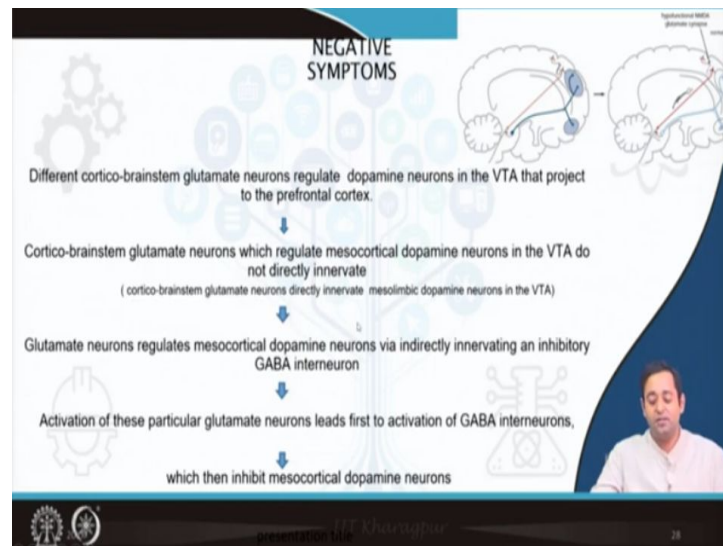
(Refer Slide Time: 19:10)



Now there is another pathway circuit which actually (res) contributes to positive symptoms. This is from hippocampus to VTA. There is a disconnect and defective hippocampal parvalbumin connecting deep GABA interneurons going to the hippocampal glutamate neuron, which is projecting to the nucleus accumbens. Then that new neuron projecting to two spiny neurons. So, this is presenting two two GABA spiny neurons. So, they both are under negative inhibition.

So, first GABA spiny neuron going from nucleus accumbens to nucleus globus pallidus from here to here. Second GABA spiny neuron going from the globus pallidus to VTA. So, you have two negative pathways which are getting cancelled off inhibited. So, they become positively. So, there is excessive firing of the neural impulses which leads to your delusions and hallucinations.

(Refer Slide Time: 20:05)



Now what is the reason behind the negative symptom? Negative symptom is caused by cortico-brainstem glutamate neurons regulating dopamine neurons in the VTA that project to the prefrontal cortex. The cortico-brainstem glutamate neurons which regulate mesocortical dopamine neurons in the VTA do not directly innervate. They do so by indirectly innervating an inhibitory GABA neuron.

So, there you go. Initially if you compare it with the positive symptoms there you have two negative pathways which were being negated. So, there you have a heightened positive neural impulse. Here you have a normal neural response which is being inhibited. So, there that is why in mesocortical area, the dopamine levels are less and which contribute to negative symptoms.

(Refer Slide Time: 20:56)

Radiological findings

- Loss of brain volume, cerebral cortex, thalamus, basal ganglia which results into reduced density of axon, dendrites and synapses from excessive pruning of synapses in adolescence.
- MRI findings suggestive of decrease in size of amygdala hippocampus para-hippocampal gyri.
- Dilatation of cerebral ventricles and reduced volume of cortical grey matter

Dr. T. Khoury

29

Radiological findings of schizophrenia, you have loss of brain volume, cerebral cortex, thalamus, basal ganglia all in reduced, leads to reduced density of axon, dendrites, which is because of excessive pruning of the synapses in adolescence. MRI findings are suggestive of size of the amygdala hippocampus. They all are decreased and there is dilation of these cerebral ventricles.

(Refer Slide Time: 21:21)

10-year MRI follow-up
34-year-old female with chronic schizophrenia

R L

Feb. 1990 1st episode Feb. 1995 5 years later Jan. 2000 10 years later

Dr. T. Khoury

30

So, you can see in this figure there is sequential dilation of the cerebral ventricles. There is atrophy of the white matter and this is observed. This is a MRI which is being followed up for a female from 34 years. So, this is a sequential increase in the size of the cerebral ventricles and there is sequentially increase in the atrophy of the soft tissues.

(Refer Slide Time: 21:46)

**ACUTE AND TRANSIENT PSYCHOTIC DISORDER
OR
BRIEF PSYCHOTIC DISORDER**

French Call it "Bouffée Delirante"

Diagnosis is appropriate when psychotic symptoms last at least 1 day but less than a month and are not associated with mood disorder, substance related disorder, or a psychotic disorder caused by general medical condition.

Onset – acute/abrupt
(abrupt < 48 hrs ; Acute within 2 weeks or less)

4 Types
1. Reactive 2. hysterical 3. stress 4. Psychogenic

F>M Age – 20-30 years

Polymorphic and unstable changing clinical picture (hallucinations, delusions) changing in type and intensity

Varying emotional state

Precipitating factors may be life events, loss of family members or friends etc

Generally has a good prognosis

APTEL

Let us talk about acute and transient psychotic disorders. Diagnosis is appropriate when psychotic symptoms last for at least 1 day and but less than a month and are not associated with mood disorders, substance related disorders, psychotic disorder or caused by a general medical condition. Onset can be abrupt within 48 hours or acute within 2 weeks or less. There are several subtypes, reactive, hysterical, stress induced and psychogenic.

Females, they are most commonly seen and age group is 20 to 30 years. Polymorphic and unstable, rapidly changing delusion and hallucinations can be delusions, can be hallucination, can be both, variation, there can be variation in the intensity also. Precipitating factors can be life events, loss of fam, family members or friends. Generally has a good prognosis. The treatment is antipsychotic for this patient.

(Refer Slide Time: 22:39)

PERSISTENT DELUSIONAL DISORDER

Delusions constitute the only conspicuous feature or the only clinical feature. (Hallucinations can be present in rare cases).

When a person exhibits delusions of atleast 1 month's duration (DSM)/3months (ICD) that cannot be attributed to other psychiatric disorders. (DSM)

(Non bizarre situations which can occur in real life)

Mean age of onset 40 years Prevalence: 0.2-0.3 % Males > females

Factors with Good Prognosis:
(Before 30 years, female sex, short duration of illness, high socio-occupational and functional adjustment, presence of precipitating factors)

Various types

- Persecutory
- Somatic
- Jealousy
- Erotomaniac
- Grandiose

Rx Antipsychotics

32

In persistent delusional disorder, you only have a can speak with picture that is delusion, which is constantly present. Here the psychosocial impairment is not present. The patient actually is freely mobile and he is capable of doing his all day-to-day life activities. Mean age of onset is 40 years. Prevalence is 0.2 to 0.3 percent and it is most commonly seen in males. Factors with good prognosis of persistent delusion disorder. Females if they are of 30 younger than 30 years, short duration of the illness. And yes, they have a high socioeconomic adjustment with precipitating factors giving rise to this psychotic process.

There are various subtypes, persecutory, somatic, jealousy, erotomania, grandiose. Persecutory when you have been followed by somebody else. Somatic illusions, when you have some delusion developing that you might have, a tumor developing inside your stomach. You have a tumor developing inside your throat or your lungs are collapsed or something is happening in your body. Jealousy is when you develop kind of jealousy towards your spouse that you have the spouse has extramarital affair.

Erotomania is when you are in love, the person is in love with somebody else who belongs from a higher socioeconomic state. And grandiose is when you have a feeling of richness. Those, heightened power you have skills which is not being acquired by somebody else.

(Refer Slide Time: 24:10)

The slide is titled "Shared Delusional Disorder". It contains the following text:

Folie Deux :
Described by French psychiatrist Lasegue and Falret in 1877

The disorder is characterised by transfer of delusions from one person to other person.
Both the person are closely associated for a long time and live together in relative isolation

Primary case- Person with delusion
Secondary case- Person who got transferred the delusion from primary case

Person in the secondary case is more often less intelligent , gullible, passive and lacks self esteem.

Folie simultanee- two persons become psychotic simultaneously

Folie a trois - 3 persons
Folie quatre - 4 persons

Early stages the delusional experience presentation time

NPTEL

33

Next is shared delusion disorder. So, shared delusion disorder is basically described by French psychiatrist. First time by French psychiatrist Lasegue and Falret in 19 88, 87, 77. This disorder is characterized by transfer of delusions from one person to another. Both the persons are closely associated for a long time and they live together in relative isolation. So, primary cases, is a person which is harboring the delusion and secondary case, which he transfers it to the other person.

So, folie simultance is when you have two persons psychotic simultaneously. Folie a tro is when 3 persons and quatre is when you have 4 persons who is harboring the delusion. Treatment for this shared delusion disorder is separation of the primary case from the secondary case and obviously antipsychotic treatment.

(Refer Slide Time: 24:55)

Schizophreniform Disorder

Sudden in onset and benign course associated with mood symptoms (mania mostly) emotional turmoil and clouding of consciousness.

Symptoms must last for atleast 1 month but less than 6 months.

Prevalence

M:F- 5:1 Life time Prevalence - 0.1 %

Relatives of patients with Schizophreniform Disorders have history of mood disorders.

Etiology and pathophysiology same as Schizophrenia.

Treatment:

Antipsychotics Carbamazepine

presentation title IT Khanna

Versus is what is schizophrenia from disorder. It is sudden in onset benign cords associated with mood symptoms, emotional turmoil and clouding of consciousness is present. Symptoms versus persist must last for at least 1 month but less than 6 months. Prevalence is 5 is to 1 and lifetime prevalence is 0.1 percent. Etiology and pathophysiology of this psychological is same as schizophrenia and treatment is antipsychotic.

(Refer Slide Time: 25:22)

Patient with Psychotic features

Consider differential diagnoses like

- Bipolar Manic Depression
- Acute and transient psychotic disorder
- Paranoid Schizophrenia
- Schizophrenia
- Severe depression with psychotic symptoms
- Mania with psychotic symptoms
- Drug induced psychosis

Establish the diagnosis of schizophrenia

Assessment

- Severity of illness
- Risk of harm to self and others
- Current substance use/dependence
- Level of functioning
- Detailed Physical examination
- Recent blood pressure, weight and observed indicated body mass index and waist circumference
- Mental Status Examination
- Investigations: Haemogram, liver function test, renal function test, fasting blood glucose test, electrocardiogram (ECG)
- Treatment history: response to previous medication trials, compliance, side effects, etc.
- Patients and caregivers beliefs about the cause of illness and beliefs about the treatment
- Assessment for social support, stigma, coping
- Assessment of caregiver function, coping and distress

Decide about treatment setting consider inpatient care in case of suicidality, severe agitation and violence, medication resistance, patient unable to care for self or the extent that therapy requires constant supervision or support, controlled general medical conditions making management difficult at the outpatient setting.

• Consult with other specialists depending on the need of the patient

Pharmacological Management

- Choose an antipsychotic based on past treatment response, past history of side effects, cost, tolerability, Patient/family preference, preferred route of administration, availability, current metabolic profile, past history of comorbidities, treatment resistance

Extrapharmacological Interventions

- Cognitive, affective, symptoms, rapid control of symptoms, suicidality, past response to ECT, augmentation etc.

Non-Pharmacological Management

- Psychoeducation
- Psychological Rehabilitation

There is no investigation Available for diagnosis of schizophrenia or any psychotic disorder as it is in any medical disorders.

Quantitative assessment is done by various scales

PANSS- Positive and Negative syndrome scale
BPRS- Brief Psychiatry rating Scale

presentation title IT Khanna

Now since there is no investigation available for schizophrenia or any other psychiatric illness, because as we see in medical disorders, you have a lot of variety of investigations to prove or to have a confirmation that this patient might be suffering from this medical disorder. Whereas, in here in psychiatry, we do not have any kind of confirmatory

investigation, which is which actually with these patient might be suffering from schizophrenia, bipolar depression.

But yes, you have a assessment. You can actually quantify the psychiatric disorder with the help of some quantitative assessment mythologies. Like here in schizophrenia or various other psychotic spectrum disorders, it can be quantified with the help of some scales that is BPRS psychiatric rating scale or PANSS that is positive and negative syndrome skill.

Now this is a chart which actually, when the patient is assessed, when the patient is being assessed and a protocol is being formed on how to get patient treated, we actually follow certain guidelines. So, patient, if at all, he is having a psychotic symptoms, so there has to be a differential diagnosis there. These are the differential, these are the queries that patient might have, patient might be suffering from. These are disorders that the patient might be suffering from. This can be the variety of other explanations, other disorders.

So, we need to rule out those other disorders. There can be organic mental conditions which can give rise to psychotic process. We have ATPD, acute transmission, psychotic disorders. You have schizophrenia, you have drugs related to psychotic conditions. You have severe depression with psychotic condition or mania with psychotic features. So, we have to get confirmed that, the psychotic disorder, which we are dealing with the patient is this. And after getting confirmation we should proceed with the illness.

So, establishment of, if we are a 100 percent sure that, the person we are dealing with is having a symptoms of schizophrenia, we should move in line of initiate the treatment in the lines of schizophrenia. So, there are various assessment that has to be done. The severity of the illness has to be assessed, the risk has to be assessed, comorbid substance abuse, if the patient is taking some kind of addiction smoking, cannabis, all these things are actually being associated with the schizophrenia.

There is detailed physical examinations to rule out any other problems with the patient might be suffering, which is actually contributing to this schizophrenia or is being actually not looked after. Mental status examination, investigations, various investigation, blood investigations that has to be done. Caregiver. So, you have to take a definite and elaborative history from the caregiver that if the patient is psychotic, he will or she will not be able to give his or her problems to you. So, for that, the caregiver has to be perfectly probed and ask what are the problem your patient is suffering from.

Assessment of the caregiver support, the coping mechanism, the skills which the patient have or the caregiver has according to this as far as the psychiatric illness is concerned. Decide about the treatment setting, whether the patient is very violent or he can be managed in the OPD. Then last is the treatment part. Treatment part consists of psychological as well as the pharmacological treatment. So, psychological treatment will be discussing in other lectures and in, pharmacological we be there are various anti-psychotic, typical atypical anti-psychotics along with benzodiazepines and anxiolytics in order to calm down the patient's agitation, aggression.

(Refer Slide Time: 29:22)

CONCLUSIONS

1. In this lecture we have learned that in order to have uniformity between treating mental health professionals two classificatory systems were formed DSM & ICD
2. Description of various psychotic disorders in brief
3. Etiology, pathophysiology of Schizophrenia
4. Reason behind positive and negative symptoms and along with treatment regimen in brief (an outline)

REFERENCES

1. Comprehensive Text Book Of Psychiatry (Kaplan & Sadock)
2. Oxford Text Book Of Psychiatry
3. Tasman & leiberman Text book of Psychiatry

So, what have you learned in this lecture? We have learned that in order to have a uniformity between treating mental health professionals, two classification systems were formed, DSM

and ICD. The description of various psychotic disorders in brief, the etiology and pathophysiology of schizophrenia and the reason behind positive and negative syndromes along with the treatment regimen in very brief. Thank you.