Basics of Mental Health and Clinical Psychiatry
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Lecture 15
Mood Disorders – 1

Hello everyone. Let us start lecture number 15, mood disorders.

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The topics we will be covering is what is mood and effect, types of mood disorders, depression etiology, its pathophysiology, treatment of depression, and various other age groups in which depression is seen that is melancholic in old age depression and atypical depression found in young age groups.

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So, what is mood? Mood is something which is defined as a pervasive and sustained emotion or feeling tone that influences a person's behaviour and colours his or her perception of being in the world. And there is a relative terminology which is used along with mood that is called affect. Affect is actually the internal representation, internal state which is being expressed outwards. And, like if, in more simplistic terms, if you try to tell this mood and affect is more commonly explained as weather and season.

Now affect is most likely taken as weather, which keeps on changing. It is the cross-sectional view of the patient mood assess, and the mood is the season, it is being related as season, which stays for a period of time, months. So, this is how we actually relate mood and affect. Now mood is when the patient is being assessed by the mental health professional or a psychiatrist, what they actually do is they have a subjective subset of symptoms which they try to correlate and see this patient is actually suffering from what kind of mood disorders.

There are various other types like depression, mania, bipolar effective disorder in which there can be mania with psychotic symptoms. You can be depression with psychotic symptoms. So, there you have unipolar depression and bipolar depression. In unipolar depression, you have depression when associated with psychosis can have psychotic manifestations like delusions, hallucinations, all sorts of things. And likewise, in bipolar effective disorder where you, where you do not have unipolar phases, you have two phases, mania as well as depression.

So, in both the cases, in both the polls you might be associated with, the patient might be associated with psychotic features. So, there we can have mania psychotic symptoms and depression with psychotic symptoms.

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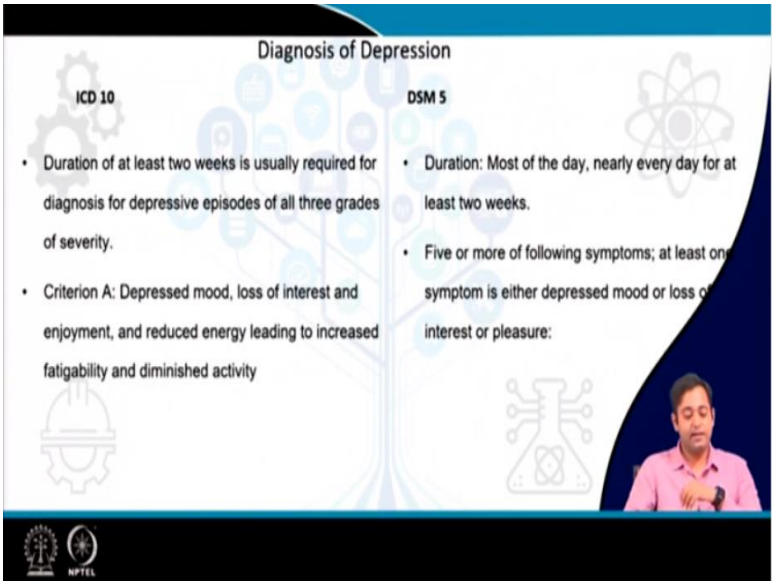

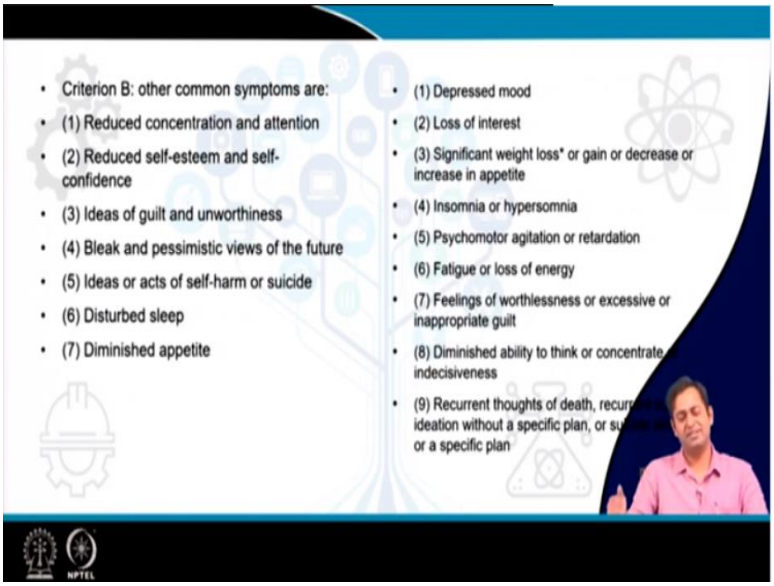

So, since there are two classificatory systems like International Classification of Disease, ICD by WHO and DSM, that is Diagnostic Statistical Manual of Mental Disorders by the American Psychiatric Association. So, they both have outlined certain set of criteria for classifying the patient certifying their patient is suffering from depression or not. Although the symptomatology and phenomenology both are same in both the classificatory systems, but the duration is a bit different and a set of criteria, they different both the classificatory systems.

So, in ICD you have at least 2 weeks of minimum duration that the symptoms should be present. Likewise, in, DSM you have 2 weeks. Now what are the main trait that in depression is looked after when the mental health professional is actually assessing the patient? It is low

mood, loss of interest that is anhedonia and fatigability. It is seen most commonly in depressed patients, which is associated with reduced concentration attention, reduced self-esteem. There might be issues of guilt, worthlessness, hopelessness, there might be decreased energy. They do not want to work also be associated with suicidal ideations. And there are some cognitive function which is decreased, like attention concentration, executive functions, they all are impaired.

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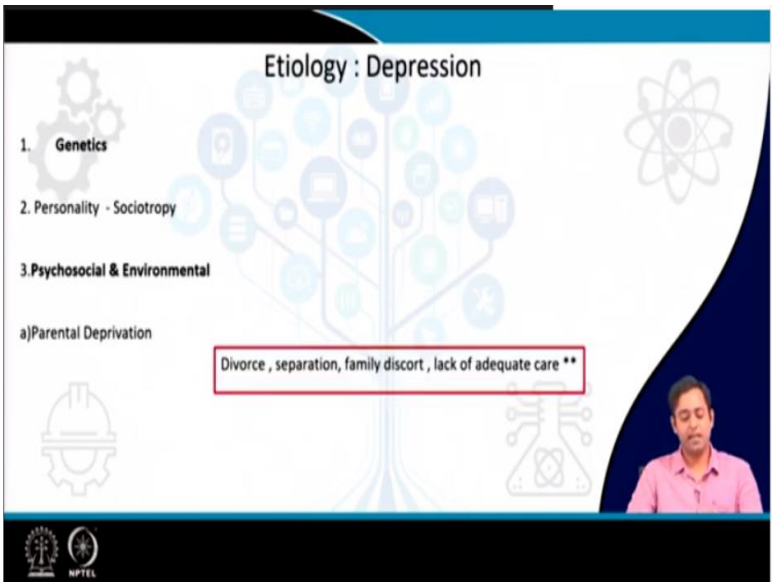

Let us look at the etiology of depression. Now, there are various causes by which depression can happen. The most important thing is that depression can also happen without any cause. That is without any etiological factor. This can happen and, during middle ages, most commonly where the person is not having any kind of stress or etiology associated, the psychosocial issues are not affected and the patient, like out of nowhere complaints that I am not feeling well, I am not able to concentrate, I am not able to enjoy my life.

Now this happens during when you are suffering from biological depression, indigenous depression without any cause. So, there are various calls. Now, what are the causes which actually the factors which leads to depression. First and foremost is genetics. In genetics, you have going to various studies which actually, which postulates that these are the factors which goes towards and causes depression.

You have twin studies available, you have family studies, you have adoption studies. There are various genes, candidate genes, which gives us an idea that this particular family, this particular gene if the family is possessing, can have, incl. There are, implications towards

going and diagnosing this patient. This patient might be suffering from depression in the family.

If you have fore fathers suffering from you have parents suffering from depression, it is not mandate or it is compulsory that you might be suffering from depression. But there are some percentage or there are some challenges, favourable chances that you might be suffering from depression. Sociotropy, personality. It is a kind of personality trait wherein the patient, actually gives more emphasis on the individual relationships rather than his personal own independence. So, that has been curtailed. His personal independence is been curtailed and he gives more emphasis rely on, rely on the relationships in which he is not intimately involved.

So, then there are psychosocial and environmental issues wherein, if the person is like, if you go back and see, if the person is having depression right now, try to elicit the history and if when assessed very properly, it has been seen that the parents, when he was child, his parents were like the marital issue of the parent, they were affected. They were, they might be having separated during the patient's childhood history. They might have a family discord in the wrong gen generation. They, the child might not be having the adequate nourishment, adequate care during his childhood days.

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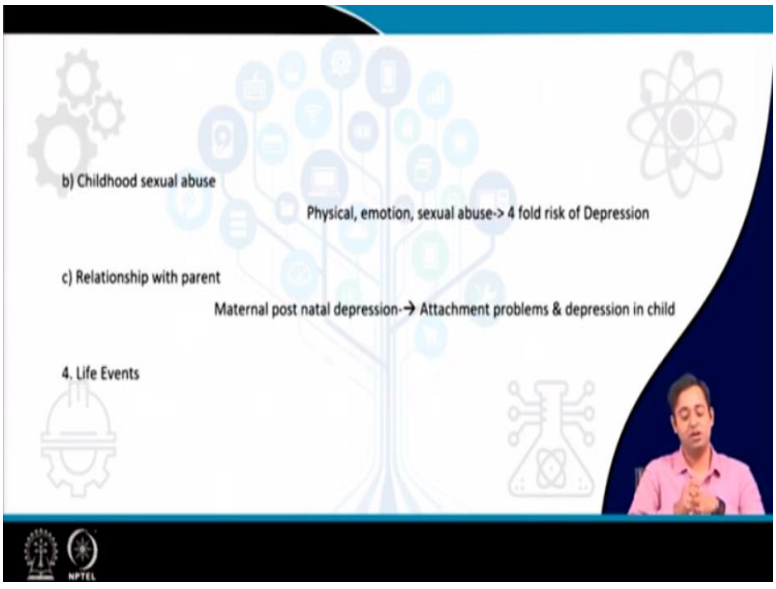

He might also be suffering from any child sexual abuse, which is the most important factor, one of the most important factors that can give rise to depression later on. There are physical emotion, sexual abuses which gives fourfold increased chances of depression. Well, what is the relationship of the parent with the person when he was child or when he is maturing into an adult?

Now, maternal post natal depression gives rise to attachment problems with the child and depression in the child also. There are life events. Life events means most important phase of the lives. When there is life-changing decisions occur. You grow, you mature into an adult, you go to college, you get married, you go to a new place, acquire a new job. During marriage, you might have a divorce when a girl goes and get married and guess to know the well like, in-laws. So, these are life-changing situations. Your spouse might die, your child might die, around 50 60 years. So, when you are not able to accommodate with the life events it can give rises to depression, these all factors they give rises to depression.

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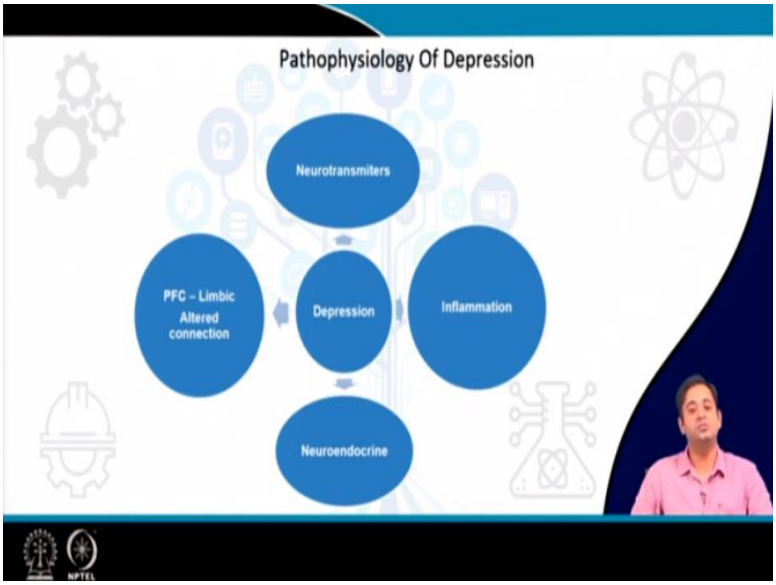

Now let us look at the pathophysiology of depression. Now, there are biological causes in which trans neurotransmitters like GABA, certalim, you have dopamine, these all are affected. You have inflammation theory wherein all sorts of cascade like interleukins and chemokines, these all are in involved. And then you have the neuro anatomical structures, which is associated and affected. That is prefrontal cortex, basal ganglia, hypothalamus, limbic areas, amygdala, these all are affected. And yes, the most important is neuroendocrine.

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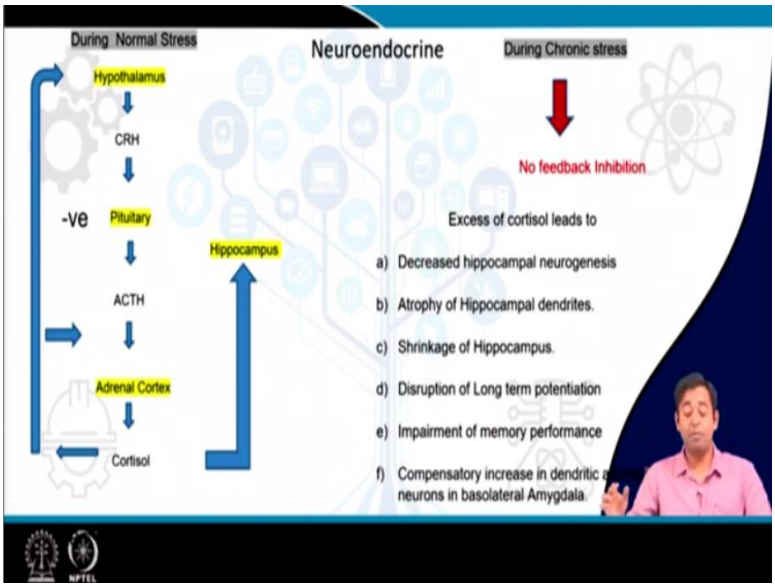

So, let us look how the neuroendocrinal changes, they give rights to they give, an important factor towards diagnosing a depression. Now, during normal stress, what happens hypothalamus, it secretes cortico releasing hormone, which gives messages to the pituitary cells and their secrete (())(9:46) hormone, which further stimulates adrenal cortex and secretes cortisol.

So, cortisol, there is a negative feedback inhibition where it gives messages to the hypothalamus, the levels of cortisol is enough to your counteract your stress levels if the patient is suffering from stress. So, this feedback loop tries to maintain the internal homeostasis of your neuroendocrine systems, your stress levels. You try to counteract with this and so does the message goes to hippocampus.

Now, what happens in chronic stress? Chronic stress, there is no feedback inhibition. This feedback inhibition is not present. And what it does, excess of cortisol is released from this adrenal cortex and it gives rise to decrease hippocampal neurogenesis. There is a trophy of the hippocampal dendrites, disruption of long-term potentiation and impairment of memory performance which leads to, and there is compensatory increasing the dendritic arborization in the bilateral amygdala.

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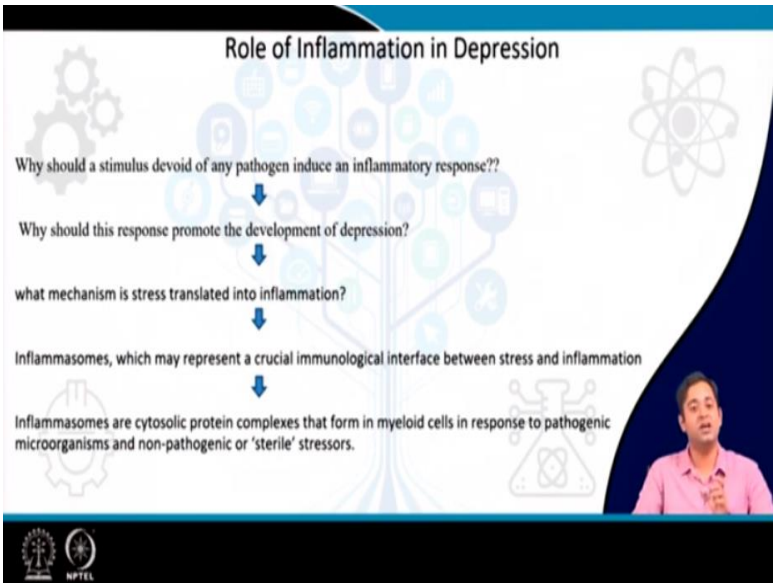

Now, does inflammation give rights to depression? Now there is a very important analogy that we should think that how should a stimulus devoid of any pathogen inducer inflammatory response. Now why should this response promote the development of depression? And what mechanism is that stress is initiating a process known as inflammation without any identifiable bacteria or virus or any other things.

So, the answer is we have inflammasome this are, they represent a crucial immunologic interface between stress and inflammation. These are a cytosolic protein complexes formed in myeloid cells in response to pathogenic microorganisms and non-pathogenic or sterile stressors. The stressors, they do not, they are not associated with any kind of microorganisms that is why they are sterile.

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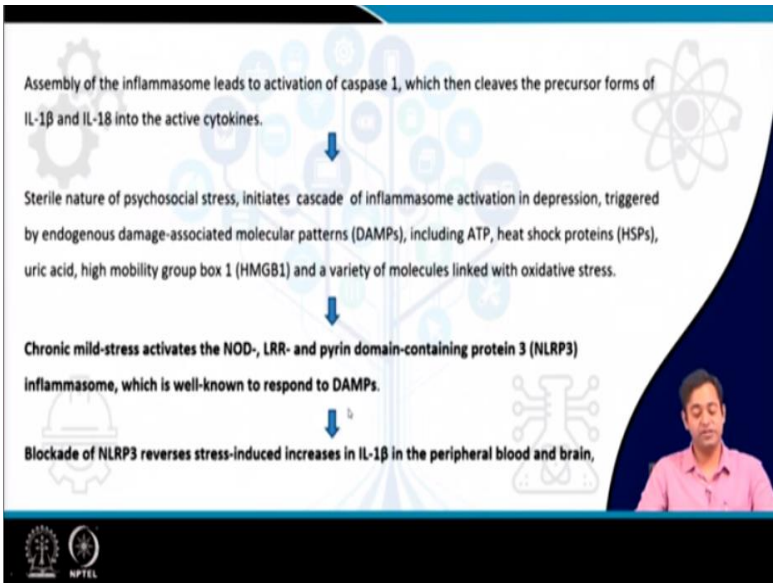

This leads to assembly of various cascade of inflammatory process of chemokims and interleukins. Sterile nature of the psychosocial stress initiates cascade of inflammasome activation in depression, triggered by endogenous damage associated molecular patterns including ATP, heat shock proteins, high mobility group box 1 index, and a variety of molecules linked with oxy oxygen oxidative stresses.

Now this pervasive stress, it activates the tall like receptors, pyrin domain containing proteins and that is how inflammation is in it is stimulated and it, which is well known to respond to damage associated proteins. Blocked of this reverses the stress induced increase in the interleukin 1 beta in the peripheral blood and brain.

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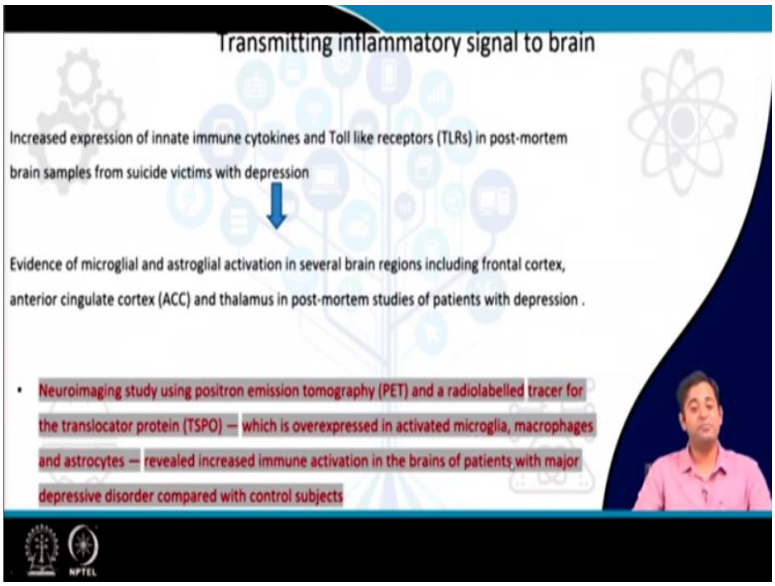

Now, there are studies available where there, they have found that positron PET scan and radio level tracer for translocated protein, which is overexpressed in activated microglia microphage astrocytes revealed increased immune activation in the brains patients with brains of patients suffering from major depressive disorder as compared to the controlled subjects.

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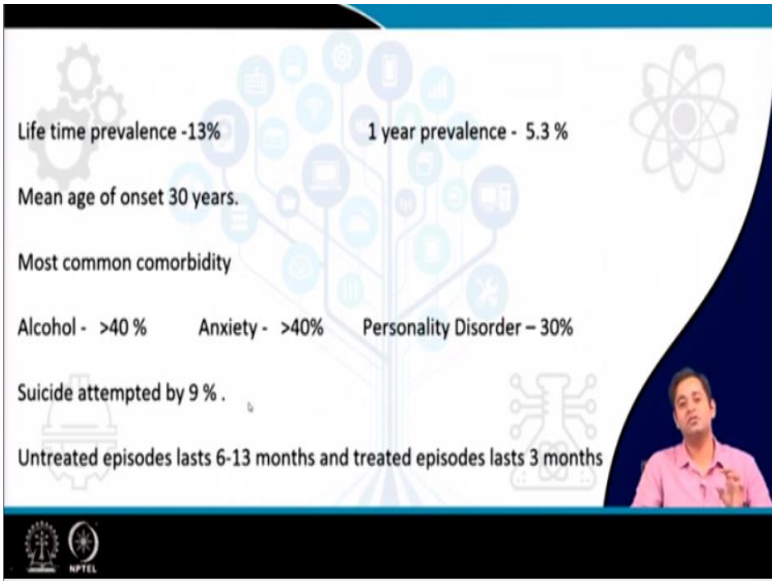

Let us look at the clinical features of patients suffering from depression. The lifetime prevalence is 13 percent. It mostly occurs during around third decade, third to fourth decade 30 years. And the most common associated disorders psychiatric illnesses with depression is alcohol because they, the persons who are suffering from alcohol, they try to self-medicate themselves by seeking substance like alcohol. They are sometimes anxious associated an anxiety disorders. And yes, personality disorders are most commonly associated with them.

Suicide attempts are 9 percent of the total population of depressed patients. An untreated depressed episode, it basically lasts for, so if you are not treating a depressive episode, it can get the, we are, if we are not treating any depressive episode, this episode can last till 6 to 13 months. And if we are treating this episode with any sort of pharmacological agent medicines, they can be treated within 3 months.

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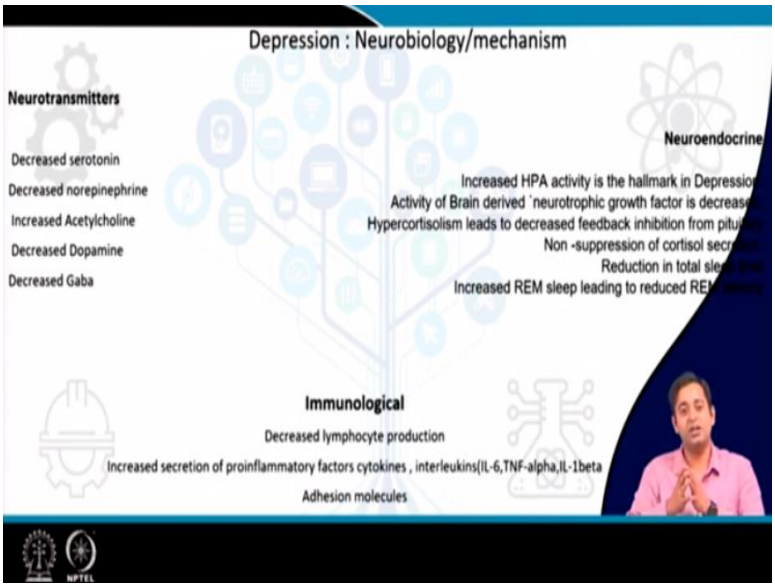

So, we have already seen the neurobiology, the pathophysiology of the depression. The neurotransmitters, which is in they are most commonly involved is serotonin nor epinephrine, acetylcholine, dopamine and GABA. These levels are decreased and acyloin levels are increased in this.

Immunologically we have seen there is decreased lymphocyte production. When the patient is suffering from depression, you might, your immunity might go down, you might be more vulnerable to all sorts of infections. There is increased submission of proinflammatory factor that is cytokines and interleukins, 1 beta adhesion molecules. And yes, there is increased HP activity, which ha which we have already seen right now in the depressed patients.

Hypercortisolism is there and there is increased REM sleep latency, which is reduced the rm like in normal sleep you have two phases and REM and rm. So, REM phase it, the is reduced. The REM most commonly occur during the later part of the sleep. So, they are actually being the sleep, the REM latency is delayed.

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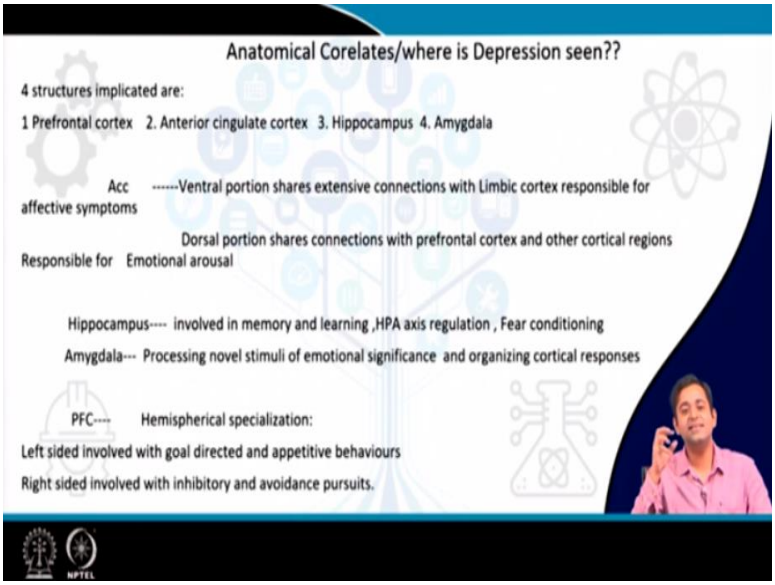

Now where is this depression occurring? What are the anatomical corelates which is associated with depression? There are 4 structures, prefrontal cortex, anterior cingulated cortex, hippocampus, and amygdala. Now anterior cingulate cortex, it shares extensive connections with limbic cortex responsible for the effective symptoms. The dorsal potion is with the emotional arousal.

Hippocampus in involved in memory and learning and amygdala is with emotional significance. Prefrontal cortex, you have left side and the right side. Left side is associated with hepatitis and goal-directed behaviours. And the right side is with the inhibitory and avoidance pursuits.

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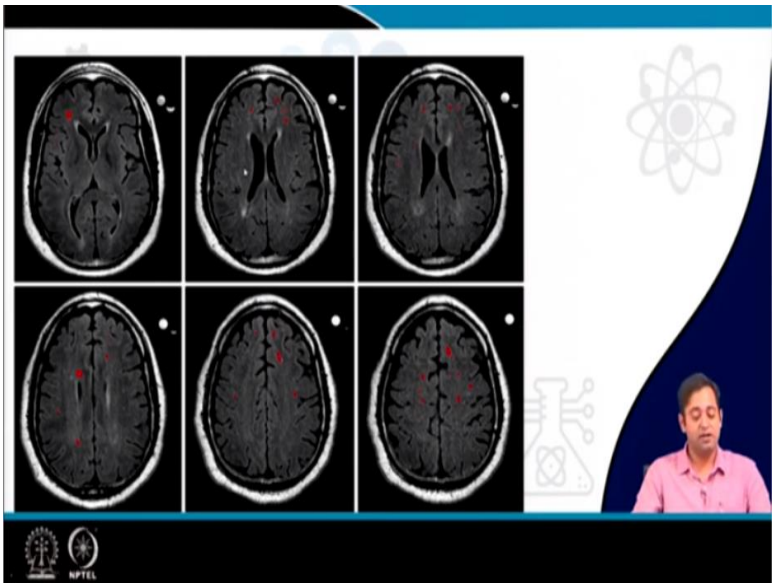

Now, structural, imaging, as you can see, this is a MRI of a depressed patient. You can see the sulcal widening of the patient as well as ventricular dilatation. And there is cortical atrophy of the person who is suffering from depression. The sulcal and the guy, they are more prominent in this person suffering from depression. Several ventricles are dilated.

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Clinical features as we have seen, there is loss of interest, pleasure, anhedonia, and they are not able to concentrate agonizing emotional pain. And they say that I am not able to enjoy my life. Two third cases, they contemplate suicide. 10 to 15 percent of the cases they commit suicide. There is reduced energy, less motivated at home and work as we have seen. There is terminal in insomnia.

Like normally when we sleep for 7 to 8 hours and when we wake up early, we ha we actually feel depressed. And, but in persons with depressed who are actually suffering from depression, there is terminal insomnia, there is early morning awakening, like usual sleep cycle of 6 to 8 hours or 7 to 8 hours is intervene. We actually get to wake up early, somewhere around 3 to 4, 4 o'clock in, in the morning. So, that is suggestive. That is one of the symptoms which is suggestive of person might be suffering from depression.

And one school of thought, which proposes that the first episode will the patient suffer, can have a long lasting changes in the brain. It can alter the functional status of the neurotransmitters and the interneuronal signaling systems.

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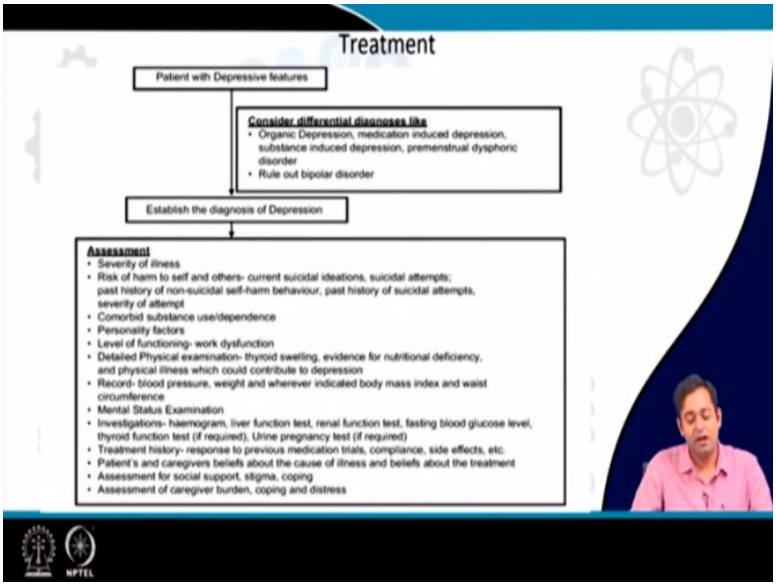

Now how can we treat this (dep) depression? We have to see what, what are the differentials? First, we have to negate the other causes that the patient might be suffering from. That is organic causes other than the psychiatric illnesses that organic is, that is dementia. You have, (())(18:07) or traumatic cases or you have, drugs or, or you have poisoning all those kind of things. You have in various sorts of infections, meningitis, encephalitis, all those has to be ruled out or substance might have produced gives given the given rise to the symptoms of depression.

So, we need to rule out all those things. Then, actually see that, we are going towards this particular person is a case of depression and the treatment should be given in the line of depression. So, the assessment has to be done in order to gauge out the severity. Is it of the mild nature, moderate nature or severe nature? What are the comorbidities that the patient

might be suffering from any kind of cardiac illnesses, any longstanding diabetes, hypertension, various comorbidities which can actually, give a picture of depression.

So, we need to treat, we have to take the help of other faculties, other, other department like cardiology, pulmonology, endocrinologists, where all those things can be sorted out.

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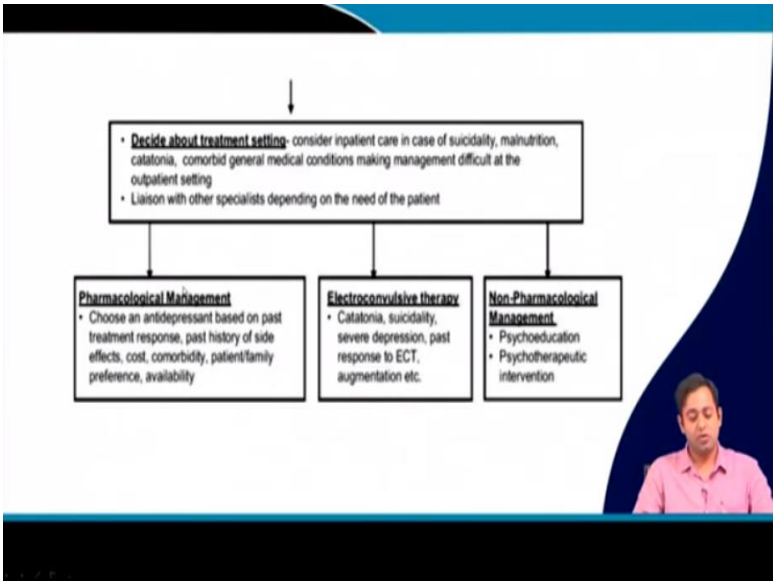

So, when we have assessed all those things, we need to decide about the treatment setting, whether this patient need OPD based treatment or they can be managed in the inpatient where we need to admit the patient in the hospital and treat them effectively. This, these can be associated with various other (con) conditions where the patient might be treated for. And there can be the management of the depression lies, pharmacologically and non-pharmacological.

Now pharmacologically, we have various different kinds of types of antidepressants. We have selective serotonin inhibitors, we have non epinephrine optic inhibitors, we have tricyclic antidepressants. We have certain mood stabilizers where we can actually give patient in order to augment the anti-depressant and leave the mood. So, there are various available options and, if you consider other modalities like, electroconvulsive therapy where the patient might need if, if the, if he or she is suffering from catatonia or if the severity is so much, so much high that, the condition has to be intervened very soon. So, ECT is commonly implicated.

If whereas, the non-pharmacological means you have psychoeducation and various sorts of psychological techniques, therapies that that can be given.

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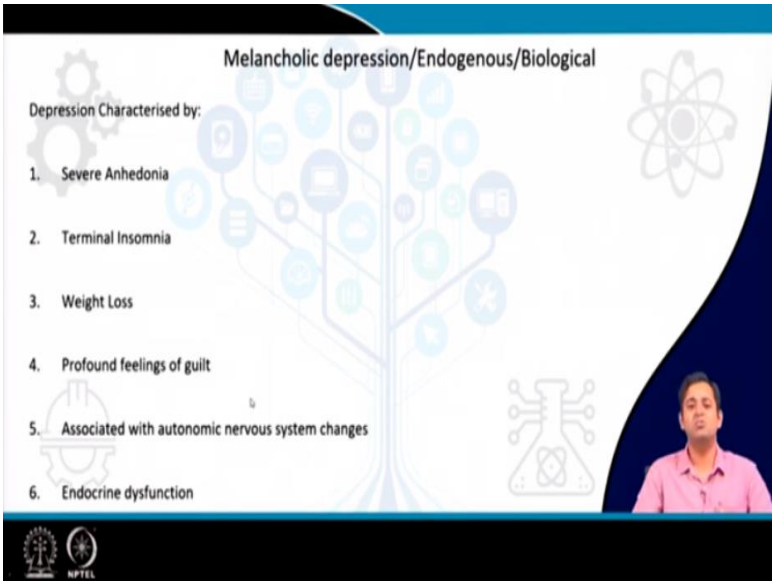

So, what are the other like, like I was telling what are the other types of presentations of depression where in different age groups, like in old ages, you this, there is something called melancholic depression, endogenous depression where there is no identifiable stressor which is being found even though the patient might be suffering from depression.

And the most common character characteristic, which is seen with melancholic depression is there is severe anhedonia, there is terminal insomnia. That is early morning awakening. There is weight loss, profound feelings of guilt. Guilt is most commonly seen with the melancholic depressed patients and they are associated with autonomic nervous changes. There is tachy cardiac, there is postural good illness, there is decrease in blood pressure. They might have perspiration sweating, which is associated with this. And yes, there is endocrine dysfunction. Various other causes, hormonal changes which might be seen in melancholic depression.

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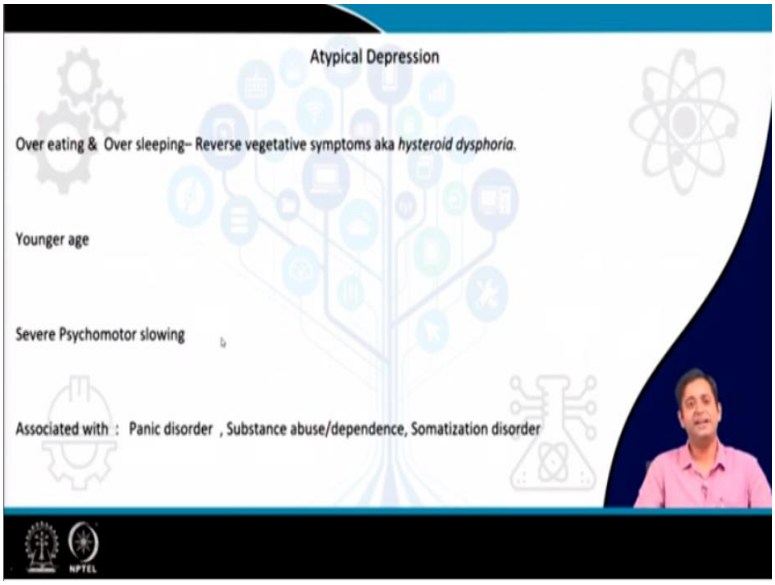

What is an atypical depression? It is otherwise known as hysteroid dysphoria. And you have this reverse vegetative symptoms like in depression, your appetite and your sleep, they get (dis) decreased. But here you have over eating and oversleeping. It is seen in younger age groups and there is severe psychomotor slowing. They are associated with panic disorders, substance abuse, dependence, and somatization.

Now what is, somatization? Somatization is when your inner psychological problems is being manifested physically. If you are suffering from any kind of stress, you are not feeling well, you re not able to express it out. So, it is being manifested physically like you might have pain somewhere in your stomach. You might have muscle spasm, muscle pain. So, these kind of things give rise to traumatization and substances when the patient is having a substance seeking behaviour. Or he might be dependent to any kind of substance like drugs, like alcohol, nicotine, you might be, addicted to cannabis opioids. There is other kinds of substances.

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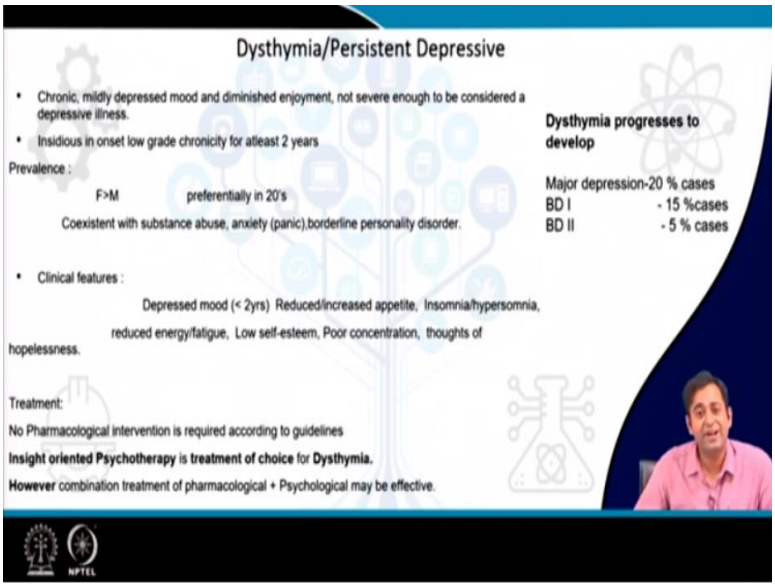

Let us look at dysthymia or what is a dysthymia? Dysthymia is chronic, mildly depressed mood and diminished enjoyment, not severe enough to be considered a depressive illness. And this if present for a period of 2 years, it is called dysthymia or persistent depressive disorder. It is commonly seen in females preferential in the age of twenties and like, all other psychiatric illnesses, they are associated with substance abuse, anxiety or borderline personality disorder.

What are the clinical features most commonly seen in case of dysthymia? It is depressed mood. Then you have reduced your increased appetite, insomnia, reduced energy, fat, fatiguability, low self-esteem, poor concentration and thoughts of hopelessness. The severity of the illness is decreased. It is lesser than the normal (())(24:02) is major depressive disorder. What is the treatment? There is no pharmacological intervention which is being required as according to the literature and various other guidelines.

The treatment of choice for this is insight oriented psychotherapy wherein the patient is given a psychotherapy, given a counselling regarding his present state of illness. And, if the patient does not get treated or does not, if the condition does not improve pharmacotherapy and the psychotherapy can be given simultaneously to uplift the status of the patient.

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Now, what is double depression? Double depression is when the dysthymia and a major depressive disorder is for a depression. They simultaneously happens for a patient. So, there is a, you can consider a patient who might be suffering from dysthymia previously from last 2-3 years of low mood, fatiguability, loss of interest, but the severity was not that much high in auto cert in order to diagnose a patient suffering from depression. So, he was actually managing day to day life activities.

His ADLs were maintained, he was doing for work, he was going for his normal social life was maintained, but suddenly he goes and lands up into a dip. He goes for land up into a depression. So, the severity and in intensity is high as compared to the dysthymia. And if this period, if this symptoms which is present for this patient for a period of 2 weeks, then we can say that this patient has actually who was suffering from dysthymia has landed up into a depression. So, this particular combined state is actually called us double depression.

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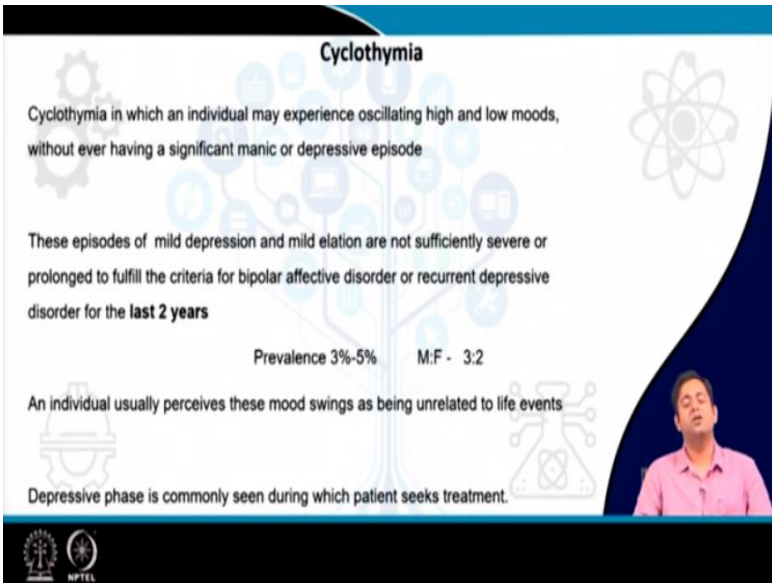

Now, likewise dysthymia, you have a condition called as cyclothymia. Now cyclothymia is a, you have oscillating highs and lows wherein the high phase that is mania and low phase that is depression. The intensity is lower than the depression and lower than the normal mania. So, you have oscillating highs and low mood without having significant manic depressive episode.

Now these episodes of mild depression and mild dilatation are not sufficiently severe to fulfil the criteria, as I have told you, for bipolar effective disorder or recurrent depressive disorder for last 2 years. And this in case of child cases, this duration goes for last 1 year. So, that is how we differentiate. Prevalence is 3 to 5 percent. And, in gender, the males are most commonly implicated. As individual usually perceives, these mood swings are being unrelated to life events and as depressive phases most commonly seen during which the patient seeks treatment.

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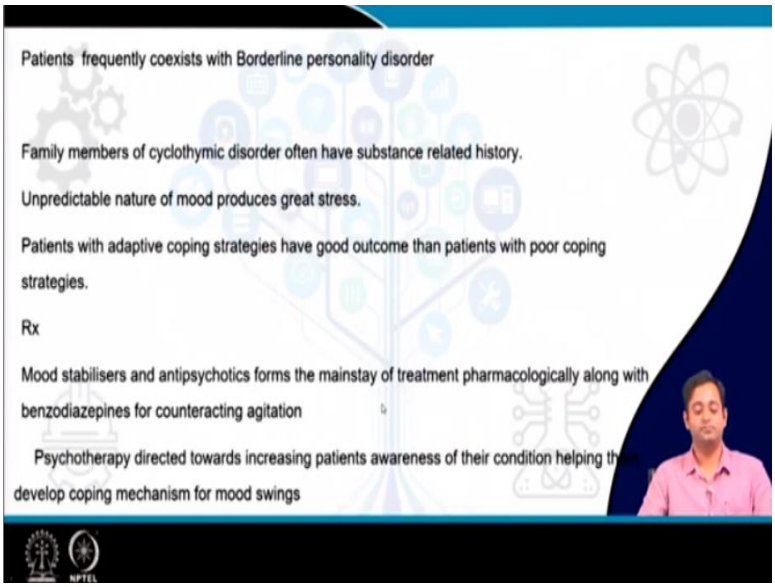

Patients frequently coexist with bipolar personality disorder. Family members of cyclothymic disorder often have substance related history. Yes, and there is unpredictable nature of the mood. This is why, mood stabilizers are commonly implicated to treat this cyclothymia. And if there is associated agitation, antipsychotics along with benzodiazepines are added in order to counteract agitation.

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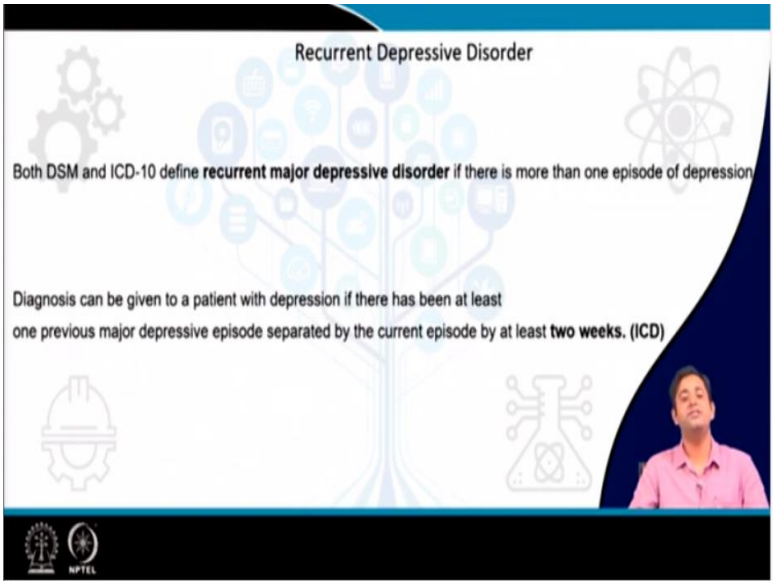

Now what is the recurrent depressive disorder? Recurrent depressive disorder is when the, you have 2 weeks, when you have 2 or more than two episodes of depressive phase and between those two episodes you have an inter episodic recovery of 2 months. And these two

depressive episodes must be there for a period of 2 weeks. So, there you go, you have 2 episodes of 2 weeks.

First episode happens for a minimum of 2 weeks. The other episode happens for a period of 2 weeks again. And in between these 2 episodes, the patient might be well for at least a period of 2 months, this is called recurrent depressive disorder, where in all those symptoms of depression is present.

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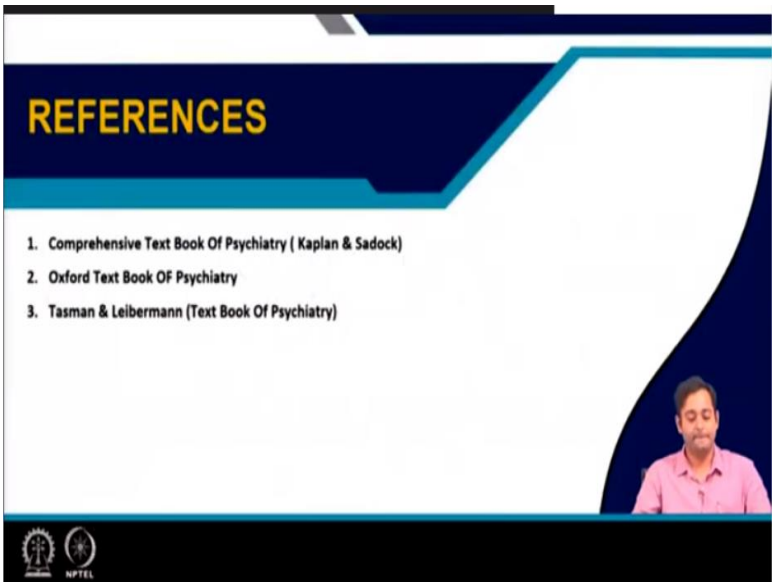

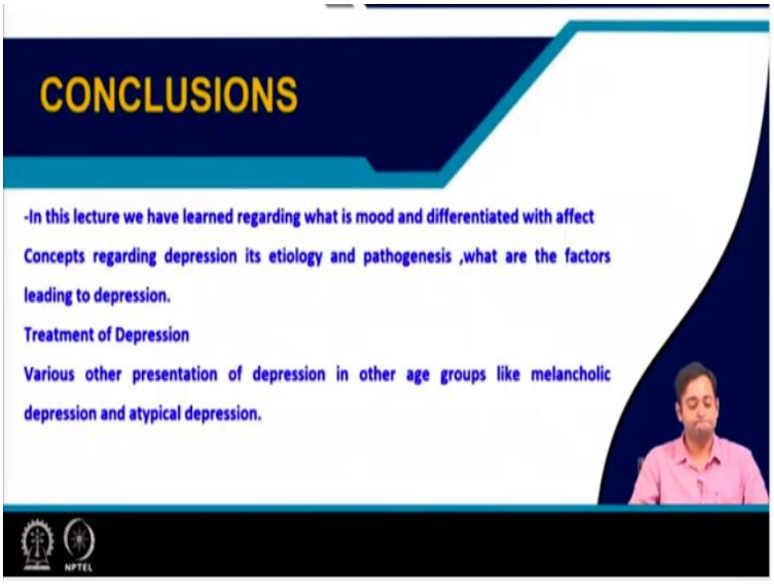

So, in this lecture, we have learned regarding what is mood, try to differentiate this, affect the concepts regarding depression, etiology, its pathophysiology, treatment, and the other presentations like dysthymia, cyclothymia and other in other age groups where depression can be seen that is old age in known as melancholic depression or atypical depression.