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Seasonal Depressive Disorder

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Continuing Education Activity

Seasonal affective disorder is a mood disorder that happens in the same season every year. It commonly occurs in young adults and women; symptoms are common in winter and fall, ranging from mild to moderate in severity. The pathophysiology is still unclear, but some data show the involvement of neurotransmitters like dopamine, norepinephrine, and glutamate. This article focuses on the importance of an interprofessional team by diagnosing and managing a patient with seasonal affective disorder.

Objectives:

- Explore the possible etiology of seasonal affective disorder.
- Review the appropriate evaluation of seasonal affective disorder.
- Summarize the management options available for seasonal affective disorder.
- Outline the importance of collaboration and communication among the interprofessional team to enhance care delivery for patients affected by seasonal affective disorder.

Access free multiple choice questions on this topic.

Introduction

Seasonal depressive disorder is a condition known to occur to a small part of the United States population. It is also called seasonal affective disorder.[1] It is dominant in areas known to experience lower sunlight levels at certain times of the year, most commonly at the change of seasons, particularly in late fall and continuing into winter, but it can also happen in the spring and summer. Symptoms related to SAD include inattentiveness, hopelessness, depression, social withdrawal, and fatigue. The symptoms have to be recurrent only during a particular time of the year for two consecutive years. It follows the recovery period after each seasonal period is over. The condition is treatable by medications, cognitive behavior therapy, and other options include vitamin D supplementation, light therapy, and electroconvulsive therapy in some cases.

Seasonal affective disorder is classified as depression related to climate and seasonal weather changes. It shares similar symptoms with other modes of depression. Most factors pertaining to the cause-and-effect mechanisms of SAD are yet to be unearthed.[1] However, several hypotheses have been put forth regarding the disease, and they show promise in delivering more information to scientists and medical personnel. For instance, a diagnosis of the disease is only made once a recurrence of the same symptoms has been established. The use of hormonal and light therapies affects an individual's circadian rhythm, hence the need for concrete proof of recurrence.

Etiology

The etiology of the condition includes several hypotheses that point to sunlight deficiency and hormonal changes as playing a role in the etiology. SAD occurs only during the climatic changes where there are significant changes in the amount of sunlight, altering the concentrations of essential hormones like serotonin and melatonin. Serotonin and melatonin regulate the body's circadian rhythm. A decrease in sunlight during winter and fall results in serotonin deficiency.[2] Apart from regulating circadian rhythm, the hormone plays a critical role in regulating mood and personality.

SAD is directly associated with elevated levels of melatonin, a common finding in most patients. A reduction in the amount of sunlight during this period enhances the levels of melatonin in the body. Cells and organs that produce melatonin are involved in regulating the circadian rhythm inducing fatigue, relaxation, and sleep when in high amounts, which generally is helpful as it occurs at night. Continued elevated levels of this hormone during the day are harmful to one’s health.

Serotonin is dependent on vitamin D availability, which is used in the active synthesis of the hormone. Vitamin D is primarily derived from sunlight. Therefore a decrease in the amount of light lowers the amount of vitamin D and, subsequently, serotonin in the blood.[3] Individuals from areas with high levels of sunlight year-round are least affected and often have optimal serotonin blood levels. Proper sleep and sufficient exposure to light are critical for SAD due to its direct effects on serotonin.

Epidemiology

In terms of physical locations, areas experiencing the four seasons seem the most affected as fluctuations in light are more common. The condition is more prevalent in young adults, especially among females. The disparity in population seems to center around young adults with peak hormone production.[2] They are often more reactive to mild changes in the environment, including sunlight, compared to older individuals. Women suffer more than men due to inactivity. Isolation from physical activities and exercises renders most females vulnerable to the condition as their hormones become either deficient or overproduced.

Individuals with a history of other mild cases of depression are more likely to contract SAD. A history of depression makes one’s body an easy target for hormonal imbalances and fluctuations. Therefore, any changes in the hormonal concentration of a particular hormone result in a vicious cycle of other hormonal imbalances leading to hormonal depressions.[4] For instance, hormonal changes affecting the release of hypothalamic hormones affect the production of stimulatory pituitary hormones, ultimately affecting hormones from the various endocrine glands.

Furthermore, the elderly can also suffer from SAD. The elderly tend to exhibit similar signs and symptoms resulting from reduced physical activity and exercise. However, changes in circadian rhythms and hormonal concentration seem to have little to no effect on SAD prevalence among the elderly.

Countries at higher altitudes experience SAD more than those at lower altitudes. Sweden experiences a significant SAD prevalence, commonly termed “winter depression.”[5] Young adults suffer this condition most frequently. Sweden has close to 8% of the cases, with twice as many women and young adults comprising those affected.[6] This incidence mostly derives from self-reported recurrent cases, and it is seasonal based on light levels. It peaks during the winter season when sunlight levels are at their lowest and less effective.[4] During spring, the depression suddenly lifts off, and an individual returns to normal.

Pathophysiology

Currently, the pathophysiology of the disease is not yet clear; researchers link the condition to available neurotransmitters. Effect on neurotransmitter availability, sensitivity, and regulation directly affect the circadian rhythms, affecting mood and leading to depression. Several neurotransmitters related to SAD include glutamate, dopamine, and norepinephrine, with others having similar effects but not to the same magnitude as this trio.[7] Several hypotheses predict evidence for a circadian phase shift associated with serotoninergic hypotheses,

but there is not yet substantial evidence to support it.[7] Although current evidence associates SAD as a biologically heterogeneous condition, it is still a hypothesis and requires more investigation to grasp the meaning.

Theories describing the pathophysiology of SAD are mainly concerned with circadian rhythm dysregulation, neurotransmitters, genetic polymorphisms, hormones, and other physiological factors. However, the serotonin hormone has the greatest effect due to its association with circadian rhythms, fatigue, and sleep induction. Serotonin is lowest in the human brain during the winter season, although it surges with increased light exposure.[8] Receptors in the brain recognize changes in the intensity of light and the duration of nights and days, inducing a reduction in the amount of serotonin secreted. Clinicians have taken advantage of this phenomenon and use light therapy to alleviate a few of the symptoms brought about by SAD.

The "phase-shift" hypothesis proposes a direct connection between the circadian rhythm and sleep-wake cycles. The current hypotheses are used in diagnosis and clinical prognosis to detect and treat SAD conditions. The endogenous circadian rhythm is known to shift during winter when day-lengths decrease and the duration of exposure and amount of light is low affecting sleep wave cycles.[9] The resultant phase-delays are proposed as the major cause of mood swings leading to SAD. Secondly, the hypotheses highlight that the body's sleep-wake cycles are phase-delayed per the dark environmental conditions. The dark cycle is associated with the increase in hours of darkness. Symptoms include delayed onset of sleep, decreased body temperature, irregular cortisol rhythms, and melatonin for some SAD patients. Therefore researchers hypothesized that the use of exogenous melatonin doses would phase-advance normal melatonin secretion in the body.

Another hypothesis entails the use of bright light to limit the body's production of melatonin. Subjects used for this experiment were winter depressives and blind individuals. They were given small doses of melatonin administered in the afternoon or evenings to initiate phase advances, while in the morning, it would induce phase delays. Both groups of individuals responded optimally to the doses given at the correct time and had their health status improve dramatically.[10] Some data suggests using melatonin level as a diagnostic tool for SAD. The research hypothesizes that elevated melatonin levels are related to a decline in SAD symptoms such as food cravings and hyperphagia. Besides, the light also affects the occurrence of SAD. Research reveals a significant association between Seasonal Pattern Assessment Questionnaire (SPAQ) results and visual impairments, which is based on SAD parameters, potentially indicating that a decrease in retinal light increases the pathogenesis of SAD. These parameters are therefore used as diagnostic and therapeutic tools.

Furthermore, the serotonin hypothesis points out that there is an association between SAD and presynaptic serotonin transporters. It mainly occurs during fall and winter, with a few outliers extending into the summer months. The monoamine hypothesis of depression points out that increased uptake of serotonin by the presynaptic neuron is dependent on the binding at the transporter—the surge results in an elevation of depressive symptoms.

Research has also linked SAD to genetic inheritance. The serotonin 2A receptor gene is closely linked to gene variation seasonality depending on the seasons; the receptor gene is highly sensitive and reactive to fluctuations in season and directly affects normal individual physiology.[11] This research was done using the Tri-dimensional Personality Questionnaire (TPQ) for credibility.[12][12] Measuring the self-rating global seasonality score against the seasonal pattern assessment helps validate the reliability of the results.[11]

History and Physical

SAD is associated with the winter season, where repetitive depression episodes often surge. The disease's existence brings about several speculations as to its existence.[13] However, taking a psychological perspective, it becomes evident that it is present. Patients suffering from SAD are prone to suicide, hence need hospitalization during such seasonal times.[14] Initially, SAD patients do not exhibit anhedonia, low mood, and other typical symptoms but rather present with other mild forms of depression.[15] These include headaches, lack of concentration, and enhanced irritability. For young people in schools, performance retardation is a typical sign, especially during the winter season.

[16]. Patients suffering from SAD are prone to suicide, hence the potential need for hospitalization during this season. [14] The elderly are most affected, with constant bouts of depressed mood.

Summer-onset of SAD tends to have distinct symptoms from the winter-phase SAD. These include insomnia, weight loss, agitation, lack of appetite, and increased irritability. The main interest is the replicability of these symptoms at the onset of their respective seasons, although with different severity. Reduced sunlight interferes with the normal body rhythm leading to the depressive symptoms mentioned above. Historically the risk of one contacting SAD is associated with younger age, female gender, family background, the presence of other depressive disorders, and living far from the equator.[17] Others suffer the depression but in a milder form and are less likely to require hospitalization.

Evaluation

The evaluation of SAD has many challenges. Treatment of SAD entails hormonal therapy and the use of neurotransmitters requiring precise diagnosis. However, SAD exhibits symptoms similar to other forms of depression. The temporal nature of mood swings also affects data acquisition. SAD is depicted as a seasonal presentation of specific depression symptoms. The pattern was observed among many Canadian youths. These variations pose a major challenge to medical personnel regarding diagnosis and therapeutic intervention.[18] Diagnosis can be made by substitution and elimination. Other major causes and history of the disease are examined and eliminated one at a time to arrive at SAD as the main cause of the depression. Physical examination is combined with a psychological evaluation to determine and discount the main cause of the depression using mechanisms such as the Patient Health Questionaire-9 (PHQ-9). It showed high levels of validity and excellent reliability for severe cases of depression.

DSM-5 is a major diagnostic criterion. It is used seasonally when the depressive symptoms are more than normal and present only in specific months of the year and are replicated at specific times.[19] The two consecutive years through which SAD diagnosis is made record two SAD episodes, which are sufficient for ascertaining the possibility of depression. However, for substantiated results, the seasonal episodes of depression should outnumber the non-seasonal episodes. Those that suffer from non-seasonal episodes will most likely report typical signs and symptoms. They mainly entail enhanced appetite, craving for carbohydrates, and increased levels of insomnia. This seasonal assessment of SAD patterns was unearthed using the SPAQ.

Treatment / Management

Being a cognitive disorder, SAD has a myriad of treatments and therapies currently in use. One of the major treatments is cognitive behavior therapy, generally once a week. Antidepressants and light therapies can accompany such treatment.[20] Other therapies include an increase in physical activities and exercise to boost dopamine elevation. Exposure to light can also be used for the regulation of melatonin and serotonin. Therefore, most individuals suffering from SAD are often advised to move to warmer places at the onset of winter and fall to prevent the above conditions. Whenever there are recurring symptoms over time, the same therapies and protocols can be used more consistently to become more effective.

The following are the main treatments used for SAD:

- Treating intrinsic medical conditions
- Light therapy
- Hormonal therapy
- Use of antidepressants
- Dietary modifications

- Increased exercises
- Meditations
- Sleep hygiene
- Dawn stimulation
- ECT therapy
- Vitamin D

With SAD as a recurring disease, the main mode of treatment involves proper planning and management. Social behavior such as drug abuse, isolation, and stress should be avoided. Activities like visiting warmer climates during winter times can aid in preventing the syndrome. Light therapy at any day or night aids in reducing depressive symptoms and advancing circadian rhythms. It can also restore sleep timing and sleep patterns. The primary form of light in light therapy is blue light, which is of a moderate frequency and normally found in the morning sunlight. It boosts vitamin D at the onset of the day. However, it should be avoided at night as it can inhibit sleeping patterns and change the physical properties of emitted light. Several devices are used to emit this type of light, although they vary greatly in the intensity and light wavelengths emitted. The efficacy of emitted light is greatly supported by research and may be useful for some patients.

Differential Diagnosis

Differential diagnosis determines the primary mode of treatment. It can reveal major depressive disorders with seasonal variations, but mild modifications have to be made to make it more specific for SAD. Other conditions that could be confused for depressive disorders include MDD, which occurs during the spring and summer months but is classified as a bipolar condition.[21] Therefore the timing of the condition is crucial to differential diagnosis.

Treatment Planning

Bright light therapy has been proven to be effective for patients of all ages, and it is also effective with depressive episodes associated with pregnancy and childbirth.[22]

Prognosis

Prognosis of SAD involves using quite a range of interventions, including antidepressants, light therapy, CBT, and vitamin D. Nearly 70% of patients experience recurring conditions of this SAD. Early diagnosis and treatment and proper education of the community on SAD are crucial in managing the condition and alleviating SAD symptoms. Close monitoring of SAD patients is also essential in managing an extreme SAD case, such as hypomania.

The cause of chorionic SAD can be either disabling or chronic, depending on the severity. Therefore, patients should first be investigated to determine the severity of the symptoms entailing the possibility of manic and hypomanic symptoms as linked to an increase in exposure to light. Patients are advised to remain compliant with their medications to avoid recurrence of the symptoms during the small window of change. Symptomatic relapse may prove more severe than the initial symptoms.

Complications

Most depressive conditions have numerous complications in treatment due to the alteration of rhythmic cycles and hormonal therapies. Secondly, they surround other depressive symptoms and include social withdrawal, lethargy and fatigue, lack of concentration, and substance abuse. Others include sadness, suicidal thoughts, headaches, weight gain, increased sleep, irritability, anxiety, and suicidal behavior.[23] Generalized anxiety disorder and other eating disorders

also induce complications, and they occur with the onset of SAD. Children exhibit signs of irritability, difficulty waking, misbehavior in school, and other related school problems.

Patients need education on real information surrounding seasonally associated disorders. They need a thorough understanding of the onset and prevention of the condition. The clinician should stress the importance of physical activity and exercise, and the patient encouraged to maintain healthy habits.[24]

Patients should be advised to move in areas with more sunlight and heat during the winter and fall months. Dietary modifications and lifestyles can also be addressed. Mechanisms through which these lifestyle modifications can be changed to alleviate a few of the conditions of SAD.

Deterrence and Patient Education

The deficiency in knowledge concerning SAD requires looking into the severity and incidence among the different groups and age brackets. It also requires differentiation from other seasonal affective disorder mimics other depressive disorders to prevent misdiagnosis.

Pearls and Other Issues

The distinction between SAD and other depressive symptoms aid is necessary to administer accurate medical intervention and exclude other possible organic causes of the depression.[25] Organic causes and syndromes should be addressed and eliminated before making a diagnosis of SAD.

Enhancing Healthcare Team Outcomes

SAD mainly affects energy output by inducing fatigue and a reduction in energy levels. It affects the daily functioning of an individual leading to lower performances and reduced productivity. The Seasonal Pattern Assessment Questionnaire (SPAQ) revealed several aspects of the condition. Therefore, much is yet to be unveiled about this form of depression. While the effects on melatonin, serotonin, and vitamin D induction from sunlight have been documented, its effects on other hormones and neurotransmitters are yet to be known. Counseling approaches are intrinsically effective in handling the condition, but supplementation with synthetic vitamin D and bright light therapy is also effective.

SAD requires the efforts of an interprofessional healthcare team that includes clinicians, mid-level practitioners, nursing staff, mental health professionals, and pharmacists. These disciplines need to exercise open communication and collaborative effort to bring about the best possible patient outcomes. [Level 5]

Review Questions

- Access free multiple choice questions on this topic.
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