Seasonal Affective Disorder

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Patients with seasonal affective disorder have episodes of major depression that tend to recur during specific times of the year, usually in winter. Like major depression, seasonal affective disorder probably is underdiagnosed in primary care settings. Although several screening instruments are available, such screening is unlikely to lead to improved outcomes without personalized and detailed attention to individual symptoms. Physicians should be aware of comorbid factors that could signal a need for further assessment. Specifically, some emerging evidence suggests that seasonal affective disorder may be associated with alcoholism and attention-deficit/hyperactivity disorder. Seasonal affective disorder often can be treated with light therapy, which appears to have a low risk of adverse effects. Light therapy is more effective if administered in the morning. It remains unclear whether light is equivalent to drug therapy, whether drug therapy can augment the effects of light therapy, or whether cognitive behavior therapy is a better treatment choice. (Am Fam Physician 2006;74:1521-24. Copyright © 2006 American Academy of Family Physicians.)
tested, the possibility of differential recall bias (depending on the time of year the test is administered), and ongoing controversy over the criteria standard for SAD. The Seasonal Pattern Assessment Questionnaire (SPAQ) is perhaps the most widely studied tool. It has been reported to have a high specificity (94 percent) for SAD but a low sensitivity (41 percent).12 Other authors, however, have reported a much lower specificity.13 The Seasonal Health Questionnaire has been reported to have higher specificity and sensitivity than the SPAQ,14 but these results must be confirmed in larger and more diverse patient groups.

Although benefits from screening are less likely to be achieved without an accurate diagnostic work-up, effective treatment interventions, and close follow-up, it is unclear whether screening ultimately improves the care and outcomes of patients with major depression. When deciding to implement a screening instrument in a practice, office personnel should consider the administration time, scoring ease, reading level, and usefulness in identifying major depression and assessing change in the depression scores over time.15

Once patients have been identified as having major depression, questions must be asked to determine if the depression is linked to SAD. These questions concern the relationship between depression and time of year (if remission occurs during certain times of the year) and whether the depression has occurred at the same time during the past two years.

Associated Diagnoses
Because SAD is associated with serotonergic dysregulation—possibly with noradrenergic mechanisms, it may overlap with other diagnoses that share similar mechanisms, including generalized anxiety disorder, panic disorder, bulimia nervosa, late luteal phase dysphoric disorder, and chronic fatigue syndrome.16 SAD also may be associated with attention-deficit/hyperactivity disorder (ADHD). Both conditions have been described as “disorders of central under-arousal coupled with a heightened sensitivity to stimuli from the physical environment,” and both are more common in women with a particular genotype for HTR2A, a gene that codes for a serotonin receptor.17,18

A pattern of seasonal alcohol use also may be associated with SAD. A summary of current research findings concluded that some patients with alcoholism may be self-medicating an underlying depression with alcohol or manifesting a seasonal pattern to alcohol-induced depression.19 Such patterns appear to have a familial component and, like the link between ADHD and SAD, may be related to serotonergic functioning.

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<th>TABLE 1 Criteria for Seasonal Pattern Specifier</th>
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<td>There has been a regular temporal relationship between the onset of major depressive episodes in bipolar I or bipolar II disorder or major depressive disorder, recurrent, and a particular time of the year (e.g., regular appearance of the major depressive episode in the fall or winter).</td>
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<td>NOTE: Do not include cases in which there is an obvious effect of seasonal-related psychosocial stressors (e.g., regularly being unemployed every winter).</td>
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<td>Full remissions (or change from depression to mania or hypomania) also occur at a characteristic time of the year (e.g., depression disappears in the spring).</td>
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<td>In the past two years, two major depressive episodes have occurred that demonstrate the temporal seasonal relationships, and no nonseasonal major depressive episodes have occurred during that same period.</td>
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<td>Seasonal major depressive episodes (as described above) substantially outnumber the nonseasonal major depressive episodes that may have occurred over the individual’s lifetime.</td>
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Treatment

Treatment options for SAD include light therapy, cognitive behavior therapy, and pharmacotherapy. Each option has been proven beneficial in treating SAD, but no large studies have found any treatment to be superior.

LIGHT THERAPY

Among susceptible persons, decreased seasonal exposure to light may mediate SAD through phase shifts in circadian rhythms, with resulting alterations in several aspects of serotonin metabolism. Thus, light replacement has been the most widely studied treatment for SAD.20 In a review of studies of light therapy, an average dosage of 2,500 lux daily for one week was superior to placebo, as indicated by improvements on a depression rating scale.21 The dosage most often found to be effective is 5,000 lux per day, given as 2,500 lux for two hours or 10,000 lux for 30 minutes.22 A recent meta-analysis of 23 studies of light therapy found that the odds ratio for remission was 2.9 (95% confidence interval, 1.6 to 5.4); this ratio is similar to those of many pharmaceutical treatments for depression.23 Like drug therapy for depression, light therapy carries some risk of precipitating mania.24

Light therapy generally is most effective when administered earlier in the day.21,25,26 Early morning light therapy regulates the circadian pattern of melatonin secretion, whereas the use of light in the evening delays the normal melatonin phase shift.27

To ensure adequate response, patients should be treated with light therapy units that are specifically designed to treat SAD. Units that are not specifically designed for SAD treatment may not provide adequate brightness and may not have appropriate ultraviolet light filtration.22

COGNITIVE BEHAVIOR THERAPY

Although cognitive behavior therapy (CBT) has some effectiveness in improving dysfunctional automatic thoughts and attitudes, behavior withdrawal, low rates of positive reinforcement, and ruminations in patients with major depression, few studies have assessed its effectiveness in the treatment of SAD. In one small clinical trial, patients with SAD were randomized to six weeks of treatment with CBT or light therapy, or CBT plus light therapy.28 At the end of treatment, all three groups had significantly decreased levels of depression, but there was no difference between groups. However, this study only enrolled 26 subjects. To date, there have been no studies large enough to establish the effectiveness of CBT in the treatment of SAD.

PHARMACOTHERAPY

Because patients with SAD also must fulfill criteria for depression, several randomized trials have assessed the use of antidepressants for this condition.29–33 Most of these studies have compared pharmacotherapy with placebo rather than light therapy, making it difficult to determine if one treatment is superior. In the largest of these trials, patients with SAD had significantly better response on several measures of depression after eight weeks of sertraline (Zoloft) therapy compared with control patients.29 Patients were excluded if they were receiving light therapy or other psychoactive medications, or if they had a history of alcoholism, drug abuse, or “emotional or intellectual problems.”

A smaller study found that, in some statistical analyses, fluoxetine (Prozac) was better than placebo in the treatment of SAD.34 Another small study found that the monoamine oxidase inhibitor moclobemide (not available in the United States) was similar to placebo in terms of changes on several general depression scales.31

Small trials of other agents (i.e., carbidopa/levodopa [Sinemet] and vitamin B12) found no benefit over placebo.34,35 Although there may be some theoretical justification for these treatments, there have not been trials of sufficient size to assess their effects.

Few randomized trials have assessed the effect of light therapy compared with pharmacotherapy.32,36 These trials failed to find a difference between the effect of 6,000 lux and that of 20 mg of fluoxetine daily,32 or between 10,000 lux and 20 mg of fluoxetine daily.36 Larger trials will be required to establish whether there is a difference in effect size between light therapy and pharmacotherapy.

It is also possible that pharmacotherapy may preserve an initial therapeutic response to light therapy. Among 168 patients who had a positive response to light therapy, citalopram (Celexa) was found to be no more effective than placebo at preventing relapse; however, it was superior in terms of some secondary measures of depression.31 In general, current evidence does not provide clear guidance as to whether antidepressant treatment is superior to light therapy, or whether antidepressants are useful as an adjunct to light therapy.

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