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Effects of Light-Therapy in Seasonal Affective Disorder – Implications for the Treatment of Patients with Cardiovascular Disease

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Several well-designed studies have given proof, that depression is linked to an excess of cardiovascular deaths. In coronary heart disease patient populations, prospective studies suggest a prognostic role for depressive disorder. The mechanisms involved in the association between depression and cardiac death are unknown, however an increased susceptibility of arrhythmias and a propensity for platelet aggregation are suspected. Seasonal affective disorder (SAD, fall-winter-depression) is a form of recurrent depressive or bipolar disorder, which is frequent in the population but surprisingly poorly recognized. Light-therapy has been proven to be a most efficacious and well-tolerated treatment for patients suffering from fall-winter-depression. In order to provide optimal care for patients with cardiac illness and to improve their prognosis, psychiatric comorbidity has to be diagnosed and vigorously treated. This review outlines the clinical features of SAD and provides guidelines for treatment. ***J Clin Basic Cardiol 2004; 7: 5–7.***

Key words: *seasonal affective disorder, winter-depression, depression, light therapy, coronary heart disease*

Psychosocial factors seem to be involved in the etiology of coronary heart disease and adversely affect the prognosis [1–3]. Numerous studies have identified and confirmed four types of risk factors:

1. Type A behavior was shown to be an independent risk factor for coronary heart disease [4]. This personality trait is characterized by hard driving and competitive behavior, a potential for hostility, pronounced impatience, and vigorous speech stylistics.
2. Psychosocial work characteristics, namely low control over work and high conflicting demands (job strain model) as well as high levels of effort combined with low perceived reward (effort-reward imbalance model) [5–7], might also contribute to a higher cardiac risk.
3. Studies investigating social network structure and quality of social support (including emotional and confiding support) in relation to the incidence of coronary heart disease were positive [8].
4. In healthy populations, prospective cohort studies showed a possible etiological role for depression and anxiety [9–11]. A dramatic increase in cardiovascular mortality in depression also remained after controlling for known cardiac risk factors such as smoking [12–14].

A number of recent studies have examined the effect of depression in clinical populations with preexisting cardiovascular disease [15, 16]. Taken together these investigations indicate in a convincing way a strong influence of depression on survival in ischemic heart disease, especially after myocardial infarction. It is surprising, that even slightly elevated scores on psychometric instruments measuring depression predict a poorer outcome in cardiac illness [17]. Among the depressed patients, who died in prospective studies, the vast majority had sudden cardiac deaths, and this would further suggest that these individuals died of arrhythmic death [18]. Furthermore many cardiac pathologies exhibit increasingly periodic behavior and a loss of variability [19]. Loss of heart rate variability has been described in depressed patients and was accounted for a low parasympathetic or a high sympathetic activity that increases the probability of ventricular fibrillation [20, 21]. Depression has also been associated with an increased susceptibility for platelet activation [22, 23], which may be a significant risk factor for ischemic heart and cerebrovascular disease. Variations in the serotonin transporter-linked promotor region (5-HTTLPR) polymorphism were shown to influence the degree of activation in depressed patients [24].

Seasonal affective disorder (SAD) is defined as a condition characterized by recurrent depressive episodes that occur annually at the same time of the year [25]. To our knowledge there are no reports about the cardiac morbidity of SAD-patients, but altered autonomic functioning in SAD has been observed [26]. In addition a disturbed serotonergic activity in the brain was found in patients suffering from SAD [27, 28] and the 5-HTTLPR polymorphism seems to influence differences of phenotype in SAD [29]. Although the results of these studies are altogether ambiguous, patients with cardiac disease should be considered at higher risk when suffering from seasonal depression. In the following paragraphs the clinical features of SAD and the possibility of treating this disorder with light therapy will be discussed.

Clinical Picture of Seasonal Affective Disorder

SAD, winter type, is a syndrome in which patients suffer from major depression during autumn or winter (fall-winter-depression) and show remission in spring or summer (Table 1). Virtually all studies to date have found a high female preponderance in this condition [30]. Onset of illness is typically in the 3rd or at the beginning of the 4th decade of life [31–36]. At the moment, diagnostic latency in Central-Europe is high: patients consult a physician familiar with the diagnosis about 10 years after the first depressive episode [37]. The disorder is following an unipolar depressive or bipolar course (with hypomanic/manic phases in spring/summer). In a large German speaking SAD-sample about 77 % of all patients were unipolar depressive, 22 % suffered from bipolar-II- and about 1 % from bipolar-I-disorder [38].

The most frequent symptoms in SAD are given in Table 2. Patients with an "atypical" symptom pattern according to

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Table 1. Criteria for seasonal pattern specifier according to DSM-IV [39]

- 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 fall or winter).
  Note: Do not include cases in which there is an obvious effect of seasonally related psychosocial stressors (e.g., regularly being unemployed in winter).
- Full remissions (or a change from depression to mania or hypomania) also occur at a characteristic time of the year (e.g., depression disappears in the spring).
- In the last 2 years, two major depressive episodes have occurred that demonstrate the temporal seasonal relationships defined in Criteria A and B, and no non-seasonal major depressive episodes have occurred during that same period.
- Seasonal major depressive episodes (as described above) substantially outnumber the non-seasonal major depressive episodes that may have occurred over the individual's lifetime.

Table 2. Psychopathology of seasonal affective disorder (frequency data taken from Winkler et al., 2002 [38])

Loss of energy, decreased activity	98.4 %
Daytime fatigue	93.7 %
Depressed mood	93.0 %
Irritability	75.1 %
Decreased libido	74.3 %
Hypersomnia	72.2 %
Carbohydrate craving	66.5 %
Anxiety	65.6 %
Increased appetite	64.6 %

Table 3. Practical guidelines for light-therapy in SAD

Intensity of light source	2,500 to 10,000 lux (measured at the distance of the patient)
Wave length	Full spectrum of visible light
Distance from light source	The patient should be seated at about 60 to 80 cm from the light source
Duration	½ to 4 hours a day (depending on the intensity of the light source); from autumn to spring on a daily basis
Daytime	Higher therapeutic effect, when light-therapy is administered early in the morning
During light therapy	Patient can be active in a seated position, but should maintain the therapeutic distance to the light source
Latency of therapeutic response	3 to 7 days until onset of antidepressant action
Non-responders	Treatment with conventional antidepressant pharmacotherapy
Partial responders	Increase duration of light therapy or combination with antidepressant medication
Side effects	Cephalea, sore eyes, irritability, eventually hypomania or suicidal tendencies

DSM-IV [39] (i.e. hypersomnia, increased appetite, weight gain, carbohydrate craving, leaden feelings in arms or legs, rejection hypersensitivity) are much more frequent than those with a "melancholic" symptom pattern (i.e. loss of pleasure, loss of affective reactivity, distinct quality of depressed mood, insomnia, decreased appetite, psychomotor agitation/retardation, inappropriate guilt). Additionally a substantial proportion of female SAD-patients suffers from premenstrual dysphoric disorder (PMDD) [40]. A large part of the general population (up to 27 %) experiences seasonal alterations of mood and behavior [41] without fulfilling the diagnostic criteria: this subsyndromal form of SAD is called s-SAD [42].

Treatment of SAD

The treatment option of choice in SAD is light-therapy, i.e. administration of bright, white full-spectrum light without any ultraviolet radiation [43] (Table 3). The effects of light-therapy are thought to be mediated exclusively through the eye [44]. The intensity of the light source should be about 10,000 lux in a distance of 60 to 80 cm. Patients should perform light-therapy on a daily basis for about a half to one hour during the season that has been associated with depressive symptoms. If patients stop the treatment during the fall/winter season depressive symptoms will return for most patients even after remission. There are contradictory results in the literature about the best daytime for light-therapy [45], but probably the antidepressant effect is higher, if treatment is performed in the morning hours [46]. During light-therapy the patient can be active in a seated position, but should be asked to maintain a therapeutic distance of about 80 cm at the most. Cheaper lamps are also available with a lower intensity of light. Patients using these lamps will have to increase duration of therapy to achieve the same therapeutic effect [47]. Onset of antidepressant action is commonly seen after three to seven days. Initially the effect of light-therapy should be measured by a clinician, e.g. in an outpatient facility, at baseline and after a treatment period of about 2 weeks. Afterwards the benefit of treatment should be assessed, before the patient is advised to buy a device for personal use.

Side-effects are rather infrequently encountered and bright-light-therapy seems to be generally well-tolerated [48]. Headache and eye-strain can occur, but rarely lead to cessation of therapy. Therapeutical light exposure has not led to ocular damage, not even over years [49]. Patients with retinal disorders should consult an ophthalmologist before light-therapy. When light-therapy is performed in the evening, some patients experience insomnia. As a complication of treatment suicidal tendencies [50] and hypomania/mania [51] have been reported in the literature.

Bright-light-therapy induces remission in 60 to 80 % of an unselected sample of SAD-patients [45]. Partial responders can increase the daily duration of light-therapy; alternatively conventional antidepressant medication can be used as an adjunctive therapy [52]. Non-responders (no benefit after two weeks) should be treated with antidepressants. For SAD-patients pharmacotherapy with SSRI (selective serotonin reuptake inhibitors) has been proven to be an effective treatment [53]. Antidepressants with a sedative potential (i.e. most of the tricyclic antidepressants) should not be the first line of treatment. Patients with a mild to moderate severity of depressive illness can alternatively benefit from hypericum [54].

Conclusions

SAD is a rather frequent psychiatric disorder in the general population (4,3–10 %) [41], but it is inadequately identified, with a diagnostic latency of about 10 years after the first depressive episode. Besides seasonal changes in mood patients also suffer from impaired social functioning and reduced performance at work [55]. SAD in its most frequent manifestation, the fall-winter-depression, is characterized in most cases by reverse vegetative symptoms such as increased appetite, carbohydrate craving, hypersomnia, daytime fatigue, and loss of energy. However, the most important clinical feature is that patients feel worst in the fall-winter months, i.e. in the time of light-deficiency. The treatment of SAD is similar to other forms of depressive disorder, except that bright-light-therapy is the treatment option of choice. Furthermore, antidepressant medication has also been shown to be effective in SAD. Depression is not a mere psychological problem, but seems to be etiologically involved in cardiac disease and interfere with the prognosis in a significant way. Therefore cardiologic therapy is well advised to take psychiatric comorbidity, e.g. depressive syndromes, into consideration.

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