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Seasonal Affective Disorder, Environmental Hypersensitivity and Somatisation

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An association between seasonal affective disorder (SAD) diagnosed according to DSM-III-R criteria and 'environmental hypersensitivity', candida hypersensitivity, and food allergies is reported in two patients. It is suggested that patients with somatisation disorder may present with symptoms of SAD and other media-popularised diagnoses in a form reminiscent of the cases of multiple media-popularised diagnoses.
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Seasonal affective disorder (SAD) is a variant of bipolar affective disorder first described by Rosenthal *et al* (1984). The syndrome is characterised by seasonal fluctuations of mood, with winter depressions and mild spring hypomania. According to DSM-III-R criteria (American Psychiatric Association, 1987), there must be three episodes of major depression occurring in the winter and two of these should have occurred in consecutive winters; in addition there should only be one episode of depression in the summer for every three years.

The depression is often associated with several atypical vegetative features, namely somnolence, fatigue, carbohydrate craving with weight gain and evening worsening of mood. Although the pathophysiology of SAD is unknown, recent hypotheses have concentrated on neuro-humoral changes (Checkley *et al*, 1989). SAD has, in recent years, attracted considerable attention in the media as a form of depression which its proponents consider common, easily treated and of predominantly organic aetiology.

Environmental hypersensitivity ('total allergy syndrome' or '20th century disease') is characterised by multiple physical symptoms, whose features often change over time. The symptoms range from mild gastrointestinal and respiratory complaints, to headache, fatigue, irritability and depression. When severe, a sufferer may be unable to lead a normal existence, believing that they are at constant risk of having life-threatening reactions to a range of common substances including foods, solvents, clothing, water and air.

Those affected may seek help from clinical ecologists who treat them with special diets, and various desensitising regimes. The entity has attracted considerable controversy in the lay and scientific literature alike, and many have questioned its validity (Brodsky, 1983; Stewart & Raskin, 1986; Stewart, 1990; Howard & Wessely, 1993).

Candida hypersensitivity syndrome is likewise associated with multiple symptoms supposedly caused by a weakening of the immune system by *Candida albicans*. Patients with this illness are generally treated with a range of antifungal medications and special diets (American Academy of Allergy and Immunology, 1986). The syndrome has likewise been criticised for its lack of scientific validity (Stewart, 1990).

Stewart & Raskin (1985) interviewed 18 patients with environmental hypersensitivity and made psychiatric diagnoses for all 18. The most common group of diagnoses were the somatoform disorders, in particular somatisation, although three patients

were schizophrenic and seven had affective disorders or anxiety states.

In a later paper, Stewart (1990) interviewed 50 consecutive cases presenting to the psychiatric services with the diagnosis of 'environmental hypersensitivity'. The main finding was that 88% of these patients were women and the median age was 39 years; 90% thought they were suffering from at least one other 'disease of fashion', of which post-infectious neuromyasthenia (or chronic fatigue syndrome), food allergy causing psychological symptoms, and candida hypersensitivity were the most common. Many patients had multiple endorsements, believing they were affected by three or more such conditions. Stewart's interpretation of this finding was that this group of patients were displaying a form of somatisation, in which psychological distress could be expressed in physical symptoms. She suggested that the attribution of their symptoms to one of these diagnoses was unsurprising in that these conditions provided explanations for their symptoms, which were endorsed by their doctors and which had a purely organic (therefore external) aetiology.

Case reports

Case 1

A 31-year-old woman was assessed for SAD. She presented with a five-year history of fatigue, low mood, and weight gain each winter, which characteristically began in October and ended in March, when she would experience a period of euphoria and was overactive. She described debilitating fatigue which prevented her from working, and hypersomnia of 14 hours per day. In retrospect she was convinced that she has had this illness most of her life.

She was also under treatment from a 'clinical ecologist' for 'environmental hypersensitivity', 'chronic candidiasis' and various food allergies. These illnesses were characterised by severe fatigue, headaches and gastrointestinal symptoms, such as nausea and bloating. They were present for some years and were said to have affected her at school.

The treatment she received consisted of sublingual, desensitising drops of six chemicals – including petroleum, phenol and cigarette smoke – which were taken at four-hourly intervals. She had alternate-day, self-administered intramuscular injections to desensitise her to casein, whey and gluten. She took glutathione complex and nystatin for her candida, as well as following a 'candida diet'.

She was resistant to any questions regarding her past. She had been adopted and knew nothing of her biological parents. Her adoptive mother was a practitioner of alternative medicine. Her school was disrupted by the illnesses; she required a taxi-cab to take her to school, and frequently missed school. Despite this she managed to attain three 'A' levels, although she had no further education. She had had two important sexual relationships and had a child from each of these liaisons. She worked as a part-time accountancy clerk when well.

On presentation she was unable to sit with her head unsupported due to the severity of her fatigue. Nonetheless, she was cheerful and garrulous at interview, when seen first in late summer. She was admitted for a trial of phototherapy in the late autumn, for one week, and at this time she complained of severe and persistent fatigue as well as low mood and anhedonia. She received 2500 lux of full-spectrum, bright, white light for three hours, twice daily. At the end of this time she reported major improvements in her level of functioning. Her score on the Modified Hamilton Rating Scale for Depression – SAD Version (SIGH-SAD; Williams *et al*, 1987) had fallen from 36 to 12. At follow-up six weeks later, the score was 6; she had been using a light box at home during this time.

Case 2

A 42-year-old married women presented with symptoms of SAD. She complained of low mood, loss of motivation, hypersomnia, increased appetite and weight gain each winter, starting in November and finishing in March. There was no history of spring hypomania. She had noted these symptoms for the past five years and had made the diagnosis of SAD herself.

In addition to these complaints she had a history of poor physical health over the past ten years. She was infertile due to pelvic inflammatory disease. She had suffered from abdominal cramps, bloating, and headaches with fatigue over this time. This had led to her seeing various alternative practitioners, including an acupuncturist, homeopath and clinical ecologist, who diagnosed 'environmental hypersensitivity' and candida hypersensitivity as well as severe premenstrual syndrome (which had left her symptom-free only two days in a month). She had tried various diets and used nystatin without any improvement in her symptoms. She was taking caprylic acid at presentation.

She was the oldest of a sibship of three and had had a painful childhood in that her father had refused to acknowledge her as he had not wanted children. She and her mother had a poor relationship. She finished school at the age of 15 and went on to study graphic design. She married at the age of 30 but had not had any children, although she had a termination of pregnancy aged 25.

When assessed she was talkative and, although there was no objective evidence of mood disturbance in her demeanour, she complained of persistent anhedonia and low mood. She scored 26 on the SIGH-SAD, however, and after a one-week trial of bright-light phototherapy this had fallen to 10. A week later when reassessed (without light therapy) the score had risen to 21.

Discussion

These two patients have several features in common. They were both well read, articulate women. They both had long histories of multiple physical symptoms (especially fatigue) and had attributed these to illnesses which have enthusiastic proponents, but are

not fully accepted in 'mainstream' medicine. They both met DSM-III-R criteria for SAD and responded to phototherapy.

There are several theoretical explanations for this association. Firstly, they could indeed be unfortunate enough to be suffering concurrently from two independent uncommon diseases (both which have fatigue as a prominent symptom). Secondly they could have been affected by genuine SAD which, as a depressive process, had caused their many physical symptoms which were then ascribed incorrectly to environmental hypersensitivity. While this is possible it is an unlikely explanation as many of the physical complaints were constant throughout the year. Finally, these women's capacity for making their own media-popularised diagnoses could have extended to SAD and the concurrence of the two syndromes is a reflection on their tendency to somatise.

In explaining this association several points seem relevant. Firstly, the vegetative features of SAD, especially fatigue, weight gain and hypersomnia, are common and relatively non-specific complaints in the general population. Furthermore, these symptoms may also be subject to seasonal variation in the general population, becoming more prominent in winter. Secondly, the media may play an important role in the way in which these patients presented. These conditions are frequently the subject of magazine articles, which often have an optimistic view of SAD as an exciting new explanation for depression. Thirdly, patients often both make the diagnosis of environmental hypersensitivity and SAD themselves, and it is common for them to request referral to specialist centres, or to refer themselves directly. Fourthly, SAD is perceived by many patients as a purely organic illness; the cause is a lack of light in the winter months – it is thus an external problem and many patients diagnosed with SAD see psychological explanations as superfluous. Finally, the illness is cured without recourse to medication due to the efficacy of light treatment.

In these respects SAD shares much with environmental hypersensitivity and the other 'diseases of fashion'. Both illnesses are characterised by common somatic symptoms for which an external cause is assumed. In both cases there is a move away from traditional medicine and the use of more 'natural' remedies. The crucial difference is that, unlike environmental hypersensitivity, there is extensive epidemiological evidence for the existence of SAD as an independent disease entity.

While these points do not invalidate the nosological status of SAD, the diagnosis must clearly be made with some caution. The fact that the chief diagnostic pointers are the timing of relapses, which in clinical practice will be judged on the retrospective and subjective reports of the patients, means that misreporting the symptoms and the history of the illness may not take an act of deliberate malingering on the part of the patient. The only way to be clear about the diagnosis is to follow the patient up in the future, but it may be both undesirable and impractical to deny phototherapy to patients in the early winter in order to validate the diagnosis. A related point is that it may be dangerous to place too much emphasis on the common and non-specific vegetative symptoms of SAD which the patient may complain of. The potential for patients with somatisation disorder to present with SAD should not be ignored.

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