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FAMILIAL REVERSE SEASONAL AFFECTIVE DISORDER - A CASE REPORT

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ABSTRACT

A patient with recurrent summer depression for seven consecutive years is described, whose mood significantly worsened with increased environmental temperature. She had a family history of recurrent summer depression in both her brother and paternal grandmother with symptoms similar to those of typical endogenous depression. The patient's mood switched to hypomania with antidepressant therapy.

KEY WORDS : Seasonal affective disorder (SAD), reverse, summer, familial, genetic, hypomania.

INTRODUCTION

Patients having seasonal affective disorders (SAD) with onset of depression in autumn or winter, have atypical symptoms of hyperphagia, hypersomnia and weight gain (Rosenthal et al, 1984). Recurrent summer depression, or the reverse seasonal affective disorder has been infrequently reported in literature, despite accounting for 35% of all cases of seasonal affective disorder as studied by Boyce and Parker (1988). The depressive symptoms of reverse SAD typically start in summer, with remission in autumn or winter. The symptoms are similar to those found in typical endogenous depression, and include anorexia, insomnia and weight loss, unlike the symptoms of winter depression. Wehr and Rosenthal (1989) observed that summer depressions are longer and more severe at low latitudes, in contrast to winter depressions, whose propensity increases with increasing latitude.

There is a paucity of literature on family history of affective disorders in patients with SAD and reverse SAD. The contribution of genetic factors in the etiology of these disorders remains unclear. Gupta (1988) reported a case of recurrent summer depression with a strong family history of bipolar disorder. A small study by Wehr et al (1987) also reported a bipolar II pattern of illness being more common in such patients.

An interesting case of recurrent summer depression that we came across, is described.

CASE REPORT

A 36 year old woman presented with her seventh episode of psychiatric illness. The current history was of 2 months of subacute onset of feeling low, accompanied by tearfulness, poor concentration and loss of interest in pleasurable activities, since the beginning of May 1994. She was irritable and complained of marked fatigue. Her self confidence declined as she was unable to do her routine household work. She felt helpless and occasionally felt it would be better if she were dead. She felt excessive restlessness, apprehension and experienced palpitations when entering warm areas like the kitchen. On the other hand, she reported distinctly feeling more relaxed and cheerful when in the shade, or sitting under a fan. She had difficulty in falling asleep, and her appetite was normal. No weight change was noticed. Her libido had decreased.

The patient had 6 consecutive simliar episodes over the past 6 years, beginning in April - May and terminating in August. She did not seek treatment for the previous episodes. There was a family history of recurrent depressive disorder in

her younger brother, occurring in summer months and resolving with the onset of autumn, for 3 consective years. There was also a history of recurrent depressive disorder in her paternal grandmother for 10 years. Both had typical endogenous depressive symptoms during their periods of illness, including morning worsening of mood, decreased sleep and decreased appetite. Both reported improvement of symptoms with cooler temperatures, with their illnesses completely resolving by autumn.

The patient had been living with her husband and children in Chandigarh for the past 10 years, after shifting from her native town of Patiala, where the climatic conditions are similar.She had no significant social, marital or financial problems. She was described to be averagely sociable, cheerful, active and responsible, though sensitive to criticism.

On examination, she was cooperative and had a sad affect. She had depressive ideation of feeling low, lacking confidence in herself, felt her condition was worsening and expressed wish to die.

Her physical examination revealed no abnormality and investigiations including a thyroid profile were within normal limits.

She was started on fluoxetione, 20 mg daily, three weeks after the onset of illness, following which her concentration and interest in household activities improved. Within ten days, she was looking cheerful and became more active. Howerer, after two weeks of fluoxetine, she was talking much louder than usual and her general rate of activity was higher than normal. she began socialising more than usual and was reported to be spending money excessively. Fluoxetine was discontinued because of the switch, and the patient was started on therapeutic lithium. With this, the patient returned to baseline functioning in two weeks, and is maintaning well on lithium prophylaxis.

DISCUSSION

The patient described is a resident of Panchkula, a satellite town of Chandigarh, situated in the Punjab plains (27° 39' to 32° 30' north latitude and 73° 51' to 77° 36' east longitude). The elevation of the Punjab plains is 693 to 1007 feet above sea level. The climate of the area is characterised by a dry hot summer from March to the end of June, and winter from middle of November to early March. The months of July, August and first half of September constitute the monsoon.

Our patient presented with a clear pattern of recurrent summer depression, the current episode switching to hypomania following antidepressant therapy. Most striking is the family history of recurrent summer depression in her brother and paternal grandmother.

Recurrent summer depression, or the reverse seasonal affective disorder has been infrequently reported in literature, compared to winter depression. A small study of 12 patients by Wehr et al (1987) reported the most common symptoms in summer depression being loss of energy, social withdrawal, anhedonia, low self - esteem, decreased talkativeness, loss of interest, oversleeping, sadness, hopelessness, guilt, suicidal thoughts and decreased libido. Interestingly, patients reported their clinical state as being influenced by temperature and humidity, with one patient improving following exposure to cold. A similar finding was present in our patient and her relatives having summer depression. Though it appears that environmental temperatures have a direct bearing on the clinical illness, controlled studies are lacking.

Boyce and Parker (1988) reported 35 % of their sample of SAD as having depressive symptoms in summer, with remission in autumn or winter. These patients also had typical endogenous depressive symptoms in contrast to the atypical symptoms of winter depressives. Gupta (1988) reported a case of severe depression in summer with hypomania or mania in winter, in a patient from North India. This patient incidentally also had a strong family history of bipolar affective disorder.

In the report described, the patient despite having 6 previous depressive episodes, switched

into hypomania only with start of antidepressant medication. This raises the issue whether recurrent summer depression may in fact be a variant of bipolar II disorder itself.

The family history of recurrent summer depression in the patient's brother and paternal grandmother points to a possible genetic basis for recurrent summer depression. This genetic propensity may mediate its effects through temperature - induced mood changes. No clear genetic studies in summer depressives have been reported to date. As such, the effect of environmental temperature on the circadian rhythm is still unclear, in contrast to the role of the photoperiod in the genesis of winter depression.

Greater work is needed to identify whether patients with antidepressant - induced switches have increased susceptibility to temperature - related mood changes. Vital information could be obtained if studies regarding seasonaliy of antidepressant - induced switches were available.

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