Correspondence

EDITED BY LOUISE HOWARD

Anorexia nervosa as a phenotype of cognitive impairment in schizophrenia

Sir: We report the case of a 14-year-old girl who first became anorexic and went on to develop a psychosis. Once the psychosis arose, her eating problems immediately disappeared.

The patient had gained weight after the age of 12 years; her height and weight were 146 cm and 59 kg (body mass index 27.7) at 13 years and 8 months of age. She gradually became concerned about her body shape. As she wished to be liked by boys of the same age, she started a diet about two months before her 14th birthday. During the next three months, she had lost 9 kg in weight. Five months after the start of the diet, she began to think that people could live without food and that she would put on weight, even if she ate a little food. Then, her menstrual cycles, which had commenced at the age of 11 years and 9 months, ceased.

She was taken by her mother, to see a paediatrician and was diagnosed as suffering from anorexia nervosa. Despite some therapeutic intervention from the paediatrician, she continued to diet and her weight decreased to 30 kg at 14 years and 6 months of age. Owing to further loss of weight (down to 24.5 kg, a 58% reduction relative to her weight before the diet) in the following 20 days, she was admitted to a local general hospital.

On admission, she still attempted to reduce weight and refused to eat. As a result, she had to be nourished intravenously. On the 14th day of admission, she developed an acute psychosis (persecutory delusions and auditory hallucinations) and, on the same day, the distorted belief that people could live without food, and the wish for further reduction of weight completely vanished and eating behaviour returned to normal.

She was treated with haloperidol for the psychosis. The psychotic symptoms, including marked thought-blocking, gradually lessened but persisted for one month. Even after improvement of the psychotic symptoms, disturbed cognition about eating and behavioural eating problems never returned. However, after the acute psychosis, mild negative features (flattened affect and social withdrawal) were noted.

There are a few reports showing that some individuals concurrently suffer from schizophrenia and anorexia nervosa (i.e. symptoms of two different disorders are superimposed) (Hsu *et al*, 1981; Ferguson & Damluji, 1988; Korkina *et al*, 1992) but no studies have been reported showing completely independent episodes of these two types of disorders (i.e. virtually no overlap of the symptoms of the two disorders) in the same individual. In this case, the time courses of eating problems and psychosis were distinct; she did not have both illnesses at the same time. It is of interest that she had a dramatic shift from a course of anorexia nervosa to a clearly schizophrenic course; it looks as if some neuronal circuits were switched off and others on. It is difficult to claim, therefore, that she suffered from two separate disorders. A more likely scenario is that the same disease process underlies the phenotypic diversities. The distorted idea of eating and body image disturbance can be attributed to the cognitive impairments resulting from the schizophrenic process. In the present case, anorexia nervosa as a clinical picture could be merely an expression of fundamental cognitive impairments that lead eventually to manifestations of psychosis.

Our observation of the dramatic change in phenotypic expressions indicates that a severe form of cognitive impairments (i.e. distorted perceptions of reality) represented by delusions and hallucinations may involve neuronal circuits that are different from those related to less severe cognitive impairments (body image disturbance) that were observed in this case and are often present in individuals with anorexia nervosa.

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De novo jet-lag psychosis

Sir: Oyewumi (1998) reported a case of schizoaffective psychosis that relapsed following jet-lag. We present two cases where typical jet-lag syndrome – fatigue, reduced concentration, and exhaustion with insomnia during local sleep time (Waterhouse *et al*, 1997) – culminated in acute *de novo* psychosis.

Case 1: Mr L., a 30-year-old man with no previous psychiatric history, was hospitalised two days after an eastward transatlantic flight with time zone change (−8 hours) evoking jet-lag disturbances – insomnia, fatigue and exhaustion – culminating in acute psychosis: elevated affect, hallucinatory behaviour, loose associations, and grandiose delusions. His Brief Psychiatric Rating Scale (BPRS) score reached 28 points.

It was decided to treat him with zuclopenthixol (10 mg/day) combined with oxazepam (20 mg/day) and melatonin (3 mg/day). The latter was added to ease the jet-lag symptoms. Within five days, the patient calmed down, his psychotic manifestations resolved, and his BPRS

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score dropped to 9 points. Soon after, he was able to fly back home escorted by his family.

Case 2: Mrs L., a 43-year-old woman with no previous psychiatric history was admitted 72 hours following an eastbound flight to Israel (−10 hours). The patient exhibited severe jet-lag manifestations culminating in florid psychotic state with elevated manic affect. Her BPRS score reached 33 points.

Treatment solely with melatonin (6 mg at bedtime) was initiated. This resulted in a good night's sleep and restoration of day/night schedule. After four nights of melatonin administration, the patient's delusional ideations resolved, and her BPRS score dropped to 8 points. She was able to fly back home unescorted. From follow-up inquiries, we learned that she did not need psychiatric treatment following her westbound return flight.

The clinical implication of this report is that apart from triggering psychotic relapse, jet-lag and changes in circadian rhythm may be associated with *de novo* psychotic breakdown. Other psychological factors, such as unfamiliar surroundings, strange language, cultural clash, and religious excitement may also play a role.

Melatonin is proposed as an agent which induces sleep and overcomes jet-lag manifestations (Arendt & Deacon, 1997; Brzezinski, 1997). Case 1 illustrates that the combined use of antipsychotic medications and melatonin served to reduce signs of psychosis and restored circadian rhythm. Case 2 suggests that melatonin may suffice in jet-lag-associated psychosis.

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Development of obsessive and depressive symptoms during risperidone treatment

Sir: Risperidone, like clozapine, has been associated with the induction or exacerbation of obsessive–compulsive symptoms, which has been hypothesised to be related to its 5-HT_2 antagonistic action (Eales & Layeni, 1994; Kopala & Honer, 1994; Remington & Adams, 1994; Alzaid & Jones, 1997). It could also be speculated that risperidone's antiserotonergic properties could lead to obsessive and depressive symptoms, as the following case demonstrates.

A 29-year-old man with ICD–10 paranoid schizophrenia was placed on risperidone monotherapy (4 mg/day). Within one month he developed ICD–10 major depression and obsessions (repetitive cursing thoughts with religious and sexual content). The depression interfered seriously with everyday activities. After starting fluoxetine (20 mg/day) his obsessions resolved within two weeks and the depression resolved within three weeks. Over the fourth week he developed akathisia, and fluoxetine was discontinued. The akathisia resolved but within the next four weeks the depressive and obsessive symptoms relapsed. Risperidone was decreased to 2 mg/day but the symptoms did not resolve (although he experienced a reduction in frequency and intensity) and the treatment was stopped. The patient was put on pimozide without re-emergence of these symptoms. He had no prior history of obsessive or depressive symptoms. There was no evidence of an organic aetiology.

Unlike many traditional antipsychotics, risperidone is a more potent antagonist of serotonin (5-HT_2) than of dopamine (D_2) receptors and this action has been postulated to contribute to its atypical effects and to produce or unmask obsessive–compulsive symptoms (Eales & Layeni, 1994; Kopala & Honer, 1994; Remington & Adams, 1994; Alzaid & Jones, 1997). There have not been any reported cases of depression or combination of depressive and obsessive symptoms in the literature up to now. The emergence of these symptoms during the course of treatment with risperidone, the positive effects of fluoxetine, the re-emergence of these symptoms after discontinuation of fluoxetine and their resolution when risperidone was discontinued, together provide strong evidence that risperidone was a causative factor.

The combination of a serotonin receptor blocker (risperidone) and a serotonin reuptake inhibitor (fluoxetine) appears to be antagonistic. It is well known that fluoxetine is useful for the treatment of depression and obsessive–compulsive symptoms. Our data suggest that serotonin blockade may underlie the development of depressive and obsessive symptoms in the course of risperidone treatment. The success of fluoxetine in treating these symptoms supports this conclusion further, as do reports involving obsessive symptoms managed with fluvoxamine (Remington & Adams, 1994) or with discontinuation of risperidone (Kopal & Honer, 1994).

However, fluoxetine caused akathisia, although it did not exacerbate the psychotic symptoms in this patient. Clinicians need to be aware of fluoxetine's potential to activate psychotic processes or cause extrapyramidal side-effects (Lindenmayer *et al*, 1990).

These topics are complicated because of the different actions of risperidone and fluoxetine on different parts of the central nervous system and on different types of receptors (5-HT_1, 5-HT_2, D_1, D_2, etc.) (Eales & Layeni, 1994; Kopala & Honer, 1994), the interactions of these two agents, as well as antidepressant effects of risperidone (Dwight *et al*, 1994).

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Definitions of depression

Sir: The naturalistic follow-up study by Ramana *et al* (1999) emphasised the

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