

Late Onset Anorexia Nervosa Treated With Olanzapine: A Case Report

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Abstract

A case of late onset anorexia nervosa (AN) treated with olanzapine is reported. The patient suffered AN onset at the age of 53 and was brought to our attention four years later in a very poor state of health due to extreme starvation and laxative abuse. She presented severe obsessions about food, a very disturbed body image, and “ascetic” rituals of self-punishment. There was no improvement of her symptoms with cognitive behavioural therapy, antidepressant drugs and inpatient nutritional therapy. After the prescription of olanzapine, the patient was more cooperative and able to maintain a stable acceptable weight, although her psychiatric and anorexic symptoms only improved partially.

Keywords: anorexia nervosa, late onset, antipsychotics, laxative abuse

INTRODUCTION

Anorexia Nervosa (AN) is generally regarded as pathology typical of adolescence. Yet late onset cases are not infrequent. According to Garfinkel & Garner (1982), approximately 3-5% of AN cases develop after 25 years of age. In the literature, the term “late onset anorexia” or “late anorexia” (Dally, 1984), is generally applied to cases of AN which start after 25 (Feighner, Robins, Guze, Woodruff, Winokur, & Munoz, 1972) or 35 (Price, Giannini, & Colella, 1985) years of age.

Some authors suggest that cases of late onset might be more frequently associated with the presence of stressful events, which precede onset and greater

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comorbidity with depression (Mynors-Wallis, Tressure, & Chee, 1992). In a retrospective study comparing a group of AN patients with onset prior to 25 years to another group with onset after that age, Boast, Coker, & Wakeling (1992) found that the only difference between the two groups was a greater weight loss in the late onset group, whereas they could not confirm the differences in comorbidity with depression. Matsumoto, Takei, Kawai, Saito, Kachi, Ohashi, Takeuchi, & Mori (2001) found a higher prevalence of extreme weight loss, laxative abuse, and self-induced vomiting in late-onset AN compared to a group of early-onset subjects.

Although late onset of the disease is considered an unfavourable predictive outcome factor (Morgan & Russell, 1975), the literature to date has reported few cases of late onset eating disorders and their treatment (Kellet, Trimble, & Thorley, 1976; Hsu & Zimmer, 1988; Beck & Andersen, 1996). The aim of the present work is to describe a case of late onset AN treated with olanzapine, an atypical new generation neuroleptic drug which has been pointed out for its beneficial effects in cases of AN (La Via, Gray, & Kaye, 2000; Mehler, Wewetzer, Schulze, Warnke, Theisen, & Dittmann, 2001; Brambilla, Segura Garcia, Fassino, Abbate Daga, Favaro, Santonastaso, Ramacciotti, Bondi, Mellado, Borriello, & Monteleone, 2007).

CASE REPORT

At the time of her first visit, G. was 57 years old. She presented the criteria for a diagnosis of a severe AN binge eating/purging type, weighed 37.4 kg, a height of 1.60 meters, and had a BMI of 14.6. She was in a very poor state of health, due to extreme starvation and regular laxative abuse.

She reported the first weight loss at the age of 14, reaching a BMI of 16.9 (starting from a BMI of 19.2) for a period of few weeks. Her second weight loss episode occurred at the age of 25, when she lost 7 kg due to gastritis to weigh 47 kg (BMI = 17.5). In the following years, her weight oscillated around 50 kg. The patient reported that her menstrual cycle was always regular up to the age of 48, when menopause began. At the age of 53, after several episodes of spontaneous rib fractures, she reported a vertebral fracture after a fall. After bed rest she lost 6 kg in two weeks (BMI = 16.6). At this weight, the patient began to manifest the symptoms of an eating disorder, displaying great attention to diet, weight, and figure. This behaviour became more pronounced at the age of 55, when G. was faced with serious difficulties as regards employment and finances. She underwent a further weight loss and was brought to our attention when she weighed 37.4 kg.

Right from the first interview, the patient showed little awareness of the disease and reported several gastrointestinal symptoms as the main cause of her dietary and psychological problems. She also displayed severe obsessive symptoms and self-punishment rituals. For these symptoms, we proposed to the patient a treatment

based on outpatient cognitive behavioural therapy and the prescription of sertraline (50 mg each morning), a type of treatment, which in some previous cases (Santonastaso, Friederici, & Favaro 2001) had led to a reduction in obsessive symptoms. A month later, the patient's weight dropped to 36.5 kg and she was admitted to a medical ward. During two hospital stays, her weight increased by a few kilos, which she regularly lost after discharge.

During her third stay in hospital, the patient underwent feeding through a nasogastric tube. At discharge, she weighed 45 kg (BMI = 17.6), the hypercatabolism indices and renal and hepatic functions were within the normal range. G. continued to suffer from a severely disturbed body image and a persistent desire to lose weight. Nevertheless, she had become more aware of the disease and she was less irritable and more cooperative. Following further weight loss, G. got down to 40 kg again, which made nutritional therapy necessary, both orally and by means of a nasogastric tube at home.

At 43 kg, both nutrition therapy through nasogastric tube and treatment with sertraline were suspended and the patient began to take olanzapine 5 mg every evening, which she tolerated well without complaining of sedation or other side effects. Twelve months after the start of this treatment, her weight was stable at around 44 kg. Her psychophysical equilibrium had improved, she appeared less obsessive about food, self-punishment rituals were less frequent, and her disturbed body image appeared slightly better. Her diet, however, is still not balanced and is inadequate as regards calorie and protein content. Spontaneous bone fractures continued to occur for some years as the result of a severe osteoporosis. On the contrary, after some years of stable weight (the patient has been followed up for 9 years after her first presentation) the bone density improved.

DISCUSSION

Few descriptions of late onset cases of AN are present in the literature. This fact could be linked to greater difficulty in diagnosing AN in older patients, due to the frequent presence of organic diseases, which could mask eating disorders or delay their diagnosis. It might also be due to the co-occurrence of other psychiatric disorders, especially major depression, which often gives rise to weight loss. In her lifetime, G. had undergone some episodes of weight loss usually associated with gastrointestinal symptoms, but there had always been a conflictual experience with her body image and a desire to lose weight. In her life, somatic complaints have often justified her abnormal dietary behaviour, which continued until the onset of the AN. Despite the fact that G. in her youth never manifested the symptoms of a full eating disorder, the attitudes and behaviour typical of AN had long been present. These dietary patterns have probably contributed to the severe osteoporosis, which seem to be present even before the onset of the episode of full

AN. The denial of the eating disorder and the presence of somatic symptoms were associated with the development of “ascetic” ideals, where going without food becomes a way of “expiating” one’s inability to cope with difficult events in life.

As regards the pharmacological aspect, the case of G. would appear to confirm both the good tolerance of olanzapine in patients with AN (La Via, Gray, & Kaye, 2000; Mehler et al., 2001; Brambilla et al., 2007), and its efficacy for improving some of the anorexic symptoms. Generally, pharmacological treatment has a limited effect on AN patients and should never be the sole form of treatment (Garfinkel & Garner, 1982; Garfinkel & Walsh, 1997; Santonastaso et al., 2001). In the past, typical neuroleptics have been used with meagre success, since obsessions with weight and body image remained very strong or even increased because of the side effect of weight gaining (Garfinkel & Walsh, 1997). Olanzapine is an atypical neuroleptic drug, which produces as a side effect a weight increase estimated at approximately between 1 – 4 kg after 6-8 weeks of treatment (La Via, Gray, & Kaye, 2000).

In the cases treated with olanzapine so far described, as well as a moderate weight increase, a reduction in the anorexic symptoms, such as a lessening of obsessive thoughts about weight and body image, was observed (La Via, Gray, & Kaye, 2000; Mehler et al. 2001; Brambilla et al., 2007). In our case, G. became more cooperative and able to accept a stable weight, but the other symptoms she displayed, such as the obsessions about food, a disturbed body image, and the presence of self-punishment rituals, showed only a partial improvement. In addition, although she underwent cognitive behavioural therapy and nutritional counselling, the quality of her food intake is still very inadequate.

Future research might be aimed at a better understanding of the natural history of late onset cases, with a view to distinguishing between cases where the disorder effectively initiates in later life and those of patients with previous episodes of the disease they have always denied. This distinction has probably some important implications in the understanding of the pathogenesis and in the planning of effective treatments.

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