ANOREXIA NERVOSA TREATMENT WITH OLANZAPINE

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Abstract

Anorexia Nervosa (AN) is a severe psychiatric disease with dangerous medical implications and potentially lethal. The medication is currently not very effective because little is known about the biological factors involved in the AN etiopathogenesis. Here, we present a study showing a strong evidence of olanzapine effectiveness in the treatment of AN. Our patient, a young woman suffering from AN, was treated for 4 months with olanzapine, an atypical antipsychotic.

After 4 months of olanzapine treatment, our patient showed a moderate increase in weight (about 2.3 kg) as well as a significative improvement of several psychopathological factors, typical of AN estimated with the scale of self-EDI. Our patient showed also a significative improvement in the typical anorexic behaviors such as the impulse to thinness, perfectionism and sense of inadequacy as well as a reduction of hostility against the therapy thus leading also an improved climate of trust between the patient, her family and medical staff.

Given the above evidences, it is reasonable to assume the use of olanzapine in the treatment of AN.

Keywords: Anorexia Nervosa, Olanzapine, Atypical Antipsychotic
Introduction

Anorexia Nervosa (AN) is a complex and severe psychiatric disease, whose etiopathogenesis is still little-known, it involves mainly young women between 15 and 20 years old (1). The AN, according to the criteria of DSM IV, is characterized by marked symptoms such as the weight loss due to the drastic reduction of the food intake, intense fear of increasing weight and to accumulate fat even though it is underweight, with or without compensating phenomena as self-induced vomiting, the use of laxatives and diuretics (2). At today, the treatment of the AN is quite uncertain (3). Many drugs have been used recently with encouraging results (4).

One of the main reasons that explain the reduced effectiveness of several drugs in controlling the AN symptoms is that all the drugs evaluated have not been sufficiently cut the possible neuro-biological implications underlying the anorexic pathology (5). In the AN disease, there are several changes in the functions of neurotransmitters and neuropeptides involved in the regulation of eating behavior, but at the moment, is not yet clear whether these disorders are the cause of the disease or if this change is a result of the marked malnutrition (6). Moreover, recently, many studies have highlighted a likely involvement of altered gene for brain neurotransmitters by causing some patients more vulnerable (7). Among the various neurotransmitters involved in controlling eating behavior of the major figures in literature relate to changes in the secretion of serotonin (5HT) and its receptors, particularly receptor 5HT-2a and 2c (8). However, the use of both tricyclic antidepressants (TCA) and the latest selective serotonin reuptake inhibitors (SSRIs), was quite disappointing. In the acute phase of the AN disease, it has been showed a changes in modulation of dopamine (DA), both with hypersecretion that hyposecretion of DA and its metabolites (9,10,11). The antipsychotics drugs of second generation, called atypical, have a profile characterized by receptor antagonism not only on receptors for dopamine D2 but also on the serotonin receptors in particular 5HT2a (5) and as a side effect the ability to induce a significative increased weight (12). The use of atypical antipsychotics, particularly the olanzapine, although in clinical trials on a small number of patients, showed an improvement in weight and in eating behavior and some psychopathological aspects such as anxiety, the obsessive and compulsive behavior, the perfectionism, the idea of thinness, the delusions about body image, hostility and loss of perception of reality (6,13,14,15,16,17). Here we report a clinical study of a young patient suffering from AN-treated with olanzapine.

Patient, Methods and Results

The anorexic patient named A.R. was a young woman (19 years old) followed by our MHU (Mental Health Unit) starting by November 2008. A.R. showed a diagnosis of AN with a marked weight loss (15 kg in two years) because of an incongruous and spontaneous calorie diet. Her clinical history showed that her anorexic symptoms were previously treated with an individual and family psychotherapy as well as with nutritional supplements and multivitamins, prescribed by family doctor without any significant clinical improvement. At the first our clinical examination, she weighed 44 kg for a height of 162 cm (BMI 17.5), with frequent episodes of self-induced vomiting and excessive physical exercise: she walked for 2/3 hours per day. Menstruation had a strong irregularity, as well as oligomenorrhea was pronounced. Blood tests were normal, although blood sugar, cholesterol and total sideremia were borderline. Also, electrolytes were normal, only amylase was slightly increased, and a slight increase in the volume of the parotid glands was also observed. Along with a simple and balanced program of nutritional rehabilitation, our anorexic patient was treated with olanzapine 2.5 mg/day for the first month and 5 mg/day for three months. At the beginning and the end of observation period, our anorexic patient has completed a psychometric test for self-eating disorders (DCA), the Eating Disorder Inventory (EDI) (18), to evaluate the size of certain psychological traits or groups of symptoms typical.
of the DCA, as the drive for thinness, bulimia ideas, dissatisfaction with their body, inadequacy, perfectionism, interpersonal conflict, fear of maturity. The control of the body weight occurred every two weeks and no side effects were observed during olanzapine treatment.

After four months of olanzapine treatment both the weight and BMI of our anorexic patient were slightly increased (see table). In addition, the EDI scores are generally improved, especially in subscale for perfectionism, the drive for thinness and a sense of inadequacy (see table).

Discussion

At today, few data report the inherent brain biochemical alterations responsible for the complex symptomatology of the AN, therefore, it is not yet possible to crop a specific drug therapy, which will impact significantly both on its symptomatic expression as well as the somatic psychopathology. Some psychopathological traits, which often are associated, as the anxiety, the obsessivity, the compulsivity, the perfectionism, the distorted perception of the body, a sense of inadequacy, interpersonal distrust, hostility and lack of insight disease, greatly limiting the possibility of intervening in anorexic patients. In our anorexic patient, olanzapine seems to act not only on the restoration of weight but also on the resistance to treatment. In fact, the use of the olanzapine has induced a significative improvement of some important psychological dimensions, as assessed through the EDI test, as well as a substantial reduction in hostility towards the family and the therapeutic approach as a whole, even apart from the slight increase weight, increasing the trust between the patient, his family and therapists.

Given the above evidences, it is reasonable to assume the use of olanzapine in the treatment of AN.

References

10. Frank GK, Bailer UF et al “Increased dopamine D2/D3 receptor binding after recovery from anorexia nervosa measured by positron emission tomography and 11-C raclopride” Biol Psychiatry 2005 58: 908-912
<table>
<thead>
<tr>
<th>Clinical Parameters</th>
<th>Before Olanzapine Treatment</th>
<th>After 4 months Olanzapine Treatment</th>
<th>Olanzapine Efficacy</th>
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<tr>
<td>Weight</td>
<td>44 kg</td>
<td>46.3 kg</td>
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<tr>
<td>BMI</td>
<td>17.5</td>
<td>18.5</td>
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<tr>
<td>EDI total</td>
<td>291</td>
<td>260</td>
<td>- 31</td>
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<tr>
<td>Stimulation of the thinness</td>
<td>42</td>
<td>34</td>
<td>- 7</td>
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<tr>
<td>Bulimia Behavior</td>
<td>30</td>
<td>29</td>
<td>- 1</td>
</tr>
<tr>
<td>Body dissatisfaction</td>
<td>44</td>
<td>41</td>
<td>- 3</td>
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<tr>
<td>Inadequacy</td>
<td>56</td>
<td>48</td>
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<td>33</td>
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<tr>
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<td>40</td>
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<td>- 2</td>
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