Treatment of bulimia nervosa with citalopram: A randomized controlled trial

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Vol. 16, No. 2 (2005-05 - 2005-08)

Biomedical Research 2005; 16 (2): 85-87

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Key words: Bulimia nervosa, serotonin, SSRI

Accepted April 07 2005

Abstract

Bulimia nervosa (BN) is one of the most frequent eating disorders in industrialized societies. Reduced serotonin activity has been suggested to trigger some of the cognitive and mood disturbances associated with BN. Therefore, pharmacological treatment of BN is mainly based on the use of selective serotonin reuptake inhibitors, that have proved effective. The biological basis of this disorder fully are not established yet.

The aim of this randomized controlled trial was to verify the efficacy of citalopram, a selective serotonin reuptake inhibitor, in a group of BN diagnosed patients.

Twenty female outpatients, with an age range of 19-28 years having BN-binge purging, as defined by the DSM IV, were assigned randomly into two treatment groups; the first group received 20-40 mg/day citalopram for 12 weeks and the second group had placebo. The study was conducted for 12 weeks with weekly clinical assessments.

At the end of the observation period, the group treated with citalopram showed a statistically significant reduction in the number of binge-eating crisis and purging with respect to the group who received the placebo only. In no case, treatment was interrupted for any emergency reasons.

This study indicates that citalopram is well tolerated and equally effective in reducing binge-eating crisis and purging in patients with BN

Introduction
The term bulimia nervosa (BN) refers to an eating behaviour characterised by episodes of convulsive greedy uncontrolled ingestion of high caloric and easy-taking food in great quantities. Compensatory behaviours to control the weight often follow these episodic crises, such as self-induced vomiting or laxatives or diuretics misusing [1-5].

Even if the bulimic patient has nearly normal weight or of slightly excess, its stability is very fragile because the idea of putting on weight becomes dominant.

In 1980 the DSM III included the BN in the official diagnostic nomenclature. In 1994 the DSM IV defined the peculiarities of the bulimic crisis (binge-eating – the term first used by Albert Stuncard in the ‘50s) regarding the quantities of food taken and patient’s lack of control on eating impulse (criterion A).

Moreover, the DSM IV identifies two subtypes: with eating behaviours (self-induced vomiting, misuse of laxatives or diuretics, etc.) and without purging behaviours, when the patient uses fasting or excessive physical exercises and no medicines or vomiting (criterion B). The first type is obviously the most serious one [1-7].

The clinical complications of BN are mainly linked to the chaos in eating, but especially to the compensatory episodes which can provoke side effects, such as the erosion of the dental enamel or the inflammation of the esophageal mucosa, up to hydro-electrolytic imbalances which can cause arrhythmia, cardiac decompensation and renal insufficiency.

The bulimic patients show nearly always mood disorders (depression), abnormal behaviours such as alcohol or drugs misusing, self-damaging behaviour, panic crisis, obsessive-compulsive symptoms, etc.

Recent theories refer to BN as a serotoninergic system disfunction. The serotonin (5HT) concentration declines in the cerebro-spinal fluid of those bulimic patients who show more binge-eating behaviours. The serotoninergic theory also explains other psychopathologic aspects which are often linked to the eating behaviour disorders [8-14].

This hypothesis possibly provokes the use of antidepression psychotropic drugs as symptomatologic therapy in BN. In recent years, selective inhibitor of serotonin’s reuptake (SSRI) is favoured as drug of choice.

Our study aims at testing the efficacy of citalopram the most selective SSRI, in symptomatologic treatment of BN.

**Materials and Methods**

The study included 20 female patients, aged between 19 and 28 who suffered from BN with purging behaviours (BN – binge purging) according to the DSM IV and the BITE scale’s diagnostic criteria. The patients, with their consent, were randomly divided into two groups of 10 women in each. The patients in the first group were given citalopram of 20 mg/day for the first week and 40 mg/day during the following seven weeks. The
patients in the second group were given placebo. The study went on for eight weeks. All patients were subjected to clinical check-up (twice-a-week for the period of 8 weeks) in order to monitor the clinical development and the possible side effects if any. The patients had maintained an accurate diary of their food choices, bulimic seizures, weight and the possible compensatory behaviours.

**Results and Discussion**

Following eight weeks of treatment, the group given citalopram showed 65% reduction in the bulimic seizures and 56% decrease in the purging behaviours. The average caloric intake was decreased into 7%, and there was a strong reduction in the glicide rate. The patients lost about 5% of their body weight (Table 1).

The second group with placebo showed 12% reduction in the bulimic seizures and 7% decrease in the purging behaviours. The average caloric intake and body weight showed no significant changes.

During the course of treatment, the following side effects were recorded:

The group given citalopram showed 38% sedation, 24% mouth dryness, 6% nausea and 3% headache.

The group which had placebo showed 9% headache, 12% light asthenia and 5% sedation (Table 1).

None of the patient went out of the trial because of serious side effects.

Our results therefore suggest that the use of citalopram in the symptomatological treatment of BN has a pronounced clinic value. The bulimic patients who were given citalopram have, indeed, showed a statistically significant decrease both in binge-eating crises and purging episodes, together with a reduced craving for carbohydrates and, even if indirectly, a modest average caloric intake, which has caused a slight weight loss at the end of the trial. Moreover, no patient treated with citalopram or placebo suffered from any drug-induced side effects. These encouraging results therefore us to suggest that the use of citalopram in the symptomatological treatment of BN is as an effective alternative to tricyclic or other SSRI.

**Table 1: The effects of citalopram vs placebo in BN**

<table>
<thead>
<tr>
<th>Clinical Effects</th>
<th>Citalopram Treated Patients (% of Reduction)</th>
<th>Placebo Treated Patients (% of Reduction)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bulimic Seizures</td>
<td>65</td>
<td>12</td>
</tr>
<tr>
<td>Purging Behaviors</td>
<td>56</td>
<td>7</td>
</tr>
<tr>
<td>Weight lost</td>
<td>5</td>
<td>Ns</td>
</tr>
<tr>
<td>Sedation</td>
<td>38</td>
<td>5</td>
</tr>
<tr>
<td>-------------------</td>
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</tr>
<tr>
<td>Mouth dryness</td>
<td>24</td>
<td>Ns</td>
</tr>
<tr>
<td>Nausea</td>
<td>6</td>
<td>Ns</td>
</tr>
<tr>
<td>Headache</td>
<td>3</td>
<td>9</td>
</tr>
</tbody>
</table>

References


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