Successful duloxetine treatment of a binge eating disorder: a case report

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Abstract

We report the successful treatment of a case of refractory binge eating disorder (BED) with duloxetine, a combined serotonin and norepinephrine reuptake inhibitor, resulting in complete remission of the patient’s bingeing behaviours. This case is discussed in the context of the existing literature on the psychopharmacology of BED. Results demonstrate that inhibition of 5-HT and noradrenaline reuptake by duloxetine markedly reduces food intake, suggesting that this may be a novel approach for the treatment of obesity.

Key words

binge eating; duloxetine; serotonin norepinephrine reuptake inhibitor (SNRI)

Introduction

Binge eating disorder (BED) is defined as a condition characterised by recurrent episodes of excessive eating but, unlike bulimia nervosa, no extreme weight control behaviours (APA, DSM-IV-TR). Although currently consigned to the ‘eating disorders not otherwise specified’ DSM-IV-TR category, recent evidence has pointed out that BED is a frequent condition. According to a study based on a nationally representative US sample, the prevalence of BED is about 3.5% in women and about 2.0% in men between the ages of 18 and 65 (Hudson, et al., 2007).

Despite the severe psychological and physical impairments such as obesity that are observed in approximately 65% of BED patients (Striegel-Moore, et al., 2001), the search for effective treatments has produced only modest advances.

Based on a review of the pharmacotherapy literature, there is limited evidence that medication and placebo differentially affect either binge eating or weight loss in patients with BED (Berkman, et al., 2006; Grilo, 2007). The anticonvulsant drug topiramate has been shown to reduce binge episodes, binge days per week, illness severity, eating-related obsessions, compulsions, body-mass index (BMI) and weight over a period of 14 weeks in placebo-controlled studies (McElroy, et al., 2003a, 2007b). Short-term placebo-controlled trials have suggested that several selective serotonin reuptake inhibitors (SSRIs), such as fluoxetine (Arnold, et al., 2002), fluvoxamine (Hudson, et al., 1998), sertraline (McElroy, et al., 2000) and citalopram, (McElroy, et al., 2003b) lead to reductions in both eating and psychiatric symptoms associated with BED. Imipramine, a tricyclic antidepressant, which primarily affects noradrenergic activity, has also been shown in a placebo-controlled trial to reduce bingeing and purging behaviours in some patients (Laederach-Hofmann, et al., 1999). However, the strength of previous research is moderate due to the relatively short length of most studies, high dropout and placebo-response rates in medication trials and lack of long-term follow-up (Yager, 2008).

There is currently no Food and Drug Administration (FDA)-approved pharmacological treatment for BED. Interestingly, sibutramine, a combined serotonin-norepinephrine reuptake inhibitor (SNRI) approved by the FDA for the long-term treatment of obesity, has been recently shown to be efficacious in producing a significant decrease in weekly binge frequency, weight, frequency of binge days and BMI. Patients have shown global improvement and increased response level, including abstinence from binge eating and reduction of BED severity as well as a reduction in psychopathological features associated with eating disorders (cognitive restraint, disinhibition and hunger) (Appolinario, et al., 2003; Wiltay, et al., 2008). Given that both SSRIs and SNRIs have shown some efficacy in the treatment of BED, and similarly, in the treatment of bulimia nervosa (Bacaltchuk and Hay, 2003), it is reasonable to hypothesise that an SNRI may provide a synergistic...
effect in the treatment of this disorder, particularly in cases which are refractory to standard treatment with SSRIs.

Duloxetine is a selective SNRI recently approved in the USA as well as in Europe for the treatment of Major Depressive Disorder. It has recently been shown to reduce symptoms of bulimia nervosa (Hazen and Fava, 2006). Animal models have demonstrated that decreased feeding observed in animals on duloxetine is most likely attributable to effects on energy intake and expenditure and that these effects are similar to those of sibutramine (Liu, et al., 2002). The properties of duloxetine, however, are generally considered to be less effective (Liu, et al., 2002; Jackson, et al., 1997b).

Here, we present a case of a male patient with treatment refractory BED who achieved remission of his bingeing behaviours only after initiating treatment with duloxetine. This occurred after the failed treatment attempts of several SSRIs, topiramate and other pharmacological treatments.

Case report

The patient is a 35-year-old single male who first presented to the outpatient psychiatry clinic at the Institute for Neuroscience, Florence (Italy) for treatment of social anxiety. He had been visited at the Institute for Neuroscience clinic.

There was no family history of psychiatric problems. As first psychiatric symptoms, the patient reported a depressive episode at the age of 23, characterised by atypical features of depression such as hyperphagia and hypersomnia. The episode resolved promptly after a combination of cognitive behavioural therapy and fluoxetine (40 mg/day), but the pattern of binge eating persisted. The patient denied any history of purging or vomiting. The episode at the age of 23, characterised by atypical features of depression, was visited at the Institute for Neuroscience clinic.

The patient is a 35-year-old single male who first presented to the outpatient psychiatry clinic at the Institute for Neuroscience clinic. The patient has been free of depressive symptoms and his social phobia appeared to be related to BED. He was objectively bingeing approximately three to four times per week and met the DSM-IV criteria for BED. The patient consistently denied purging episodes. The diagnosis was determined at the time of screening using the BED diagnostic version of the Eating Disorder Examination, which is an investigator-based, semi-structured interview designed to assess the disorder (Fairburn and Cooper, 1993). Measures include the frequency of binge days (i.e., mean days/week when the patient binges once or more), weight and BMI. Quality of life was measured by the Impact of Weight on Quality of Life-Lite total score (Kolotkin, et al., 2001), and subjective distress related to body weight was measured by the Sheehan Disability Scale (Sheehan, 1983). The patient received a 5–10-min session of dietary/lifestyle psychoeducation and was first treated with fluoxetine at a dose of up to 60 mg/day over a 2-month period. The patient was visited every 2 weeks. During each visit, the time, content, ratings of loss of control and distress related to binge episodes were recorded. He initially demonstrated a favourable response to fluoxetine, losing 2 kg during the first month and a reduction in binge episodes to once a week. The following month he immediately returned to his starting weight. Following treatment with fluoxetine, the patient underwent several unsuccessful medication trials over a period of 3 years. A 4-week trial of fluvoxamine (up to 400 mg/day) had no effect on his bingeing behaviours. Trials of citalopram (80 mg/day for 4 weeks), imipramine (150 mg/day for 8 weeks) and desipramine, which is a tricyclic antidepressant that acts selectively on noradrenaline reuptake (150 mg/day for 12 weeks), were also ineffective. A 6-week trial with reboxetine (12 mg/day), a noradrenergic agent, also failed to work. When treated with topiramate (250 mg/day), the patient showed little improvement, and even topiramate given with fluoxetine for 4 months had no effect. The treatment was discontinued due to cognitive impairment and paresthesias. Atomoxetine, given its reported efficacy on BED in a randomised-controlled trial (McElroy, et al., 2007a), was attempted but quickly discontinued due to constipation and jitteriness after 2 weeks. Finally, no changes were observed when the patient took the dopamine reuptake inhibitor bupropion (400 mg/day for 8 weeks) (Horne, et al., 1988).

Following the unsuccessful trials described above, the patient was started on duloxetine at an initial dose of 60 mg/day. The medication was well tolerated and the dose was increased up to 120 mg/day over a period of 2 weeks. After approximately 1 month of treatment, the patient started losing a significant amount of weight (5 kg ending the first month) and the frequency of his bingeing episodes started reducing significantly (binge episodes frequencies reduced of 50% or more). This benefit has persisted and his condition has improved; he has now been free of bingeing behaviours for approximately 24 weeks and has lost 25 kg. On the Clinical Global Impression-Improvement scale (Guy, 1976), the patient’s score reduced from 5 to 1. The Sheehan Disability Scale scores changed from 8 to 4, from 10 to 2 and from 7 to 5, respectively, on work, social life and family life disability impacts. The Impact of Weight on Quality of Life-Lite score changed from 72 to 43 with a particular improvement on public distress domain.

Implication for clinical care

In summary, we describe a patient with severe BED whose symptoms were refractory to treatment with multiple medications, including trials of several SSRIs. The patient had a favourable response to treatment with duloxetine, a selective serotonin and norepinephrine reuptake inhibitor. To our knowledge, this is the first report of the successful use of an SNRI in a
case of BED. Our results are consistent with previous success using duloxetine for the treatment of refractory bulimia nervosa (Hazen and Fava, 2006). As in that report, it should be noted that the dose of duloxetine used to treat the patient in this study (120 mg/day) is above the recommended dose range for the treatment of depression (40–60 mg/day).

In addition to reducing the frequency of binge eating episodes, duloxetine treatment also significantly decreased food intake compared to treatments with selective serotonin, noradrenalin and dopamine agents such as citalopram, desipramine, reboxetine and bupropion. Duloxetine has been shown to reduce food intake in food-deprived rats (Katoh, et al., 1995) and in freely feeding rats during the dark phase (Jackson, et al., 1997a,b). Duloxetine also shows a thermogenic property, although it is weaker than sibutramine, but stronger than buproprion and fluoxetine (Liu, et al., 2002). Preclinical evidence in general suggests that serotonergic and noradrenergic agents act strongly on reducing food intake compared with selective serotonergic or noradrenergic agents. The effect of duloxetine on food intake has also been reported to be weaker than that for sibutramine over a short-time period. Duloxetine, however, is a more manageable agent compared with sibutramine in terms of side effects and may be the solution in BED cases in which cardiovascular risks are present (Wernicke, et al., 2007). Cardiovascular risks are often associated with obesity and in this condition sibutramine use is contraindicated.

Treatments with sibutramine at the effective dose oblige the clinician to carefully monitor blood pressure and modify treatment accordingly. Clinicians have raised concerns about the side effects of the long term use of sibutramine. An increase in blood pressure is a common side effect of every agent with noradrenergic properties. Since duloxetine reduces food intake with less α-receptor stimulation, this agent also seems to be more manageable with regard to cardiac and blood pressure side effects (Wernicke, et al., 2007).

Further research is warranted on the specific mechanism of action of duloxetine and its use in the treatment of eating disorders. This case report may set the basis for further controlled studies assessing the efficacy of duloxetine in BED.

Disclosure of commercial and non-commercial interests

The authors have no commercial and non commercial interest involved in this report to discuss.

References


