

Venlafaxine Treatment of Binge-Eating Disorder Associated With Obesity: A Series of 35 Patients

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Background: Binge-eating disorder is a newly recognized eating disorder characterized by recurrent episodes of binge eating without extreme weight loss behaviors. It commonly co-occurs with overweight and obesity. To preliminarily explore the effectiveness and tolerability of venlafaxine in binge-eating disorder, we retrospectively reviewed the response of 35 consecutive overweight or obese outpatients with binge-eating disorder presenting at the University of Cincinnati Physicians Weight Management Program, Cincinnati, Ohio, to clinical treatment with venlafaxine.

Method: The medical charts of 35 consecutive outpatients with binge-eating disorder (DSM-IV criteria) and overweight (body mass index [BMI] = 25.0–29.9) or obesity (BMI \geq 30.0) who received clinical treatment with venlafaxine at a weight management program were reviewed. Response of binge-eating disorder symptoms was assessed by weekly binge frequency (the number of binges reported by the patient the week before the clinic appointment), the Clinical Global Impressions-Severity of Illness (CGI-S) scale, and categorical response (no response, mild, moderate, marked, or remission). Weight, BMI, waist circumference, comorbid Axis I diagnoses, vital signs, and side effects also were collected.

Results: Twenty-nine patients (83%) received venlafaxine as monotherapy and 6 (17%) received the drug adjunctively for a median of 120 days (range, 28–300 days). The mean \pm SD venlafaxine treatment dose was 222 \pm 63 mg/day (range, 75–300 mg/day). In the 33 patients who were actively binge eating at the time venlafaxine was begun, weekly binge frequency, severity of binge-eating and mood symptoms as measured by the CGI-S scale, weight, BMI, waist circumference, and diastolic blood pressure all showed statistically significant decreases over time ($p < .05$). Of these 33 patients, 29 (88%) displayed a moderate (50% reduction) or better response of binge-eating episodes. Fifteen (43%) of the 35 patients lost 5% or more of their baseline weight. In general, venlafaxine was well tolerated, with dry mouth, sexual dysfunction, insomnia, and nausea being the most frequently reported side effects. Sustained increases in blood pressure seen in 6 patients (17%) were considered clinically insignificant. No patients discontinued the drug.

Conclusion: Venlafaxine may be an effective treatment for binge-eating disorder associated with overweight or obesity. Controlled studies of venlafaxine in binge-eating disorder appear warranted.

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Binge-eating disorder is a newly recognized eating disorder characterized by recurrent episodes of binge eating without extreme weight loss behaviors.^{1–5} It is associated with overweight and obesity and is particularly common among persons seeking help for obesity.^{1–11} Approximately 30% of obese individuals in standard weight loss treatment programs, 70% of those in Overeaters Anonymous, and up to 50% of those seeking bariatric surgery may suffer from binge-eating disorder.^{2,6,7,9,10} It may also be common among the general population, with the prevalence in males being perhaps two thirds that in females.^{2,5–8}

At present there is no established psychopharmacologic treatment for binge-eating disorder.^{3,5,12} Two recently completed double-blind, placebo-controlled studies, however, suggest that binge-eating disorder,^{13,14} like bulimia nervosa,^{15–17} may respond to treatment with selective serotonin reuptake inhibitors (SSRIs). In the first study,¹³ fluvoxamine (50–300 mg/day) was significantly superior to placebo in reducing binge frequency and body mass index (BMI) in a 9-week trial. In the second study,¹⁴ sertraline (50–200 mg/day) was associated with significantly greater reduction in the frequency of binges, clinical global severity, and BMI and a significantly greater rate of clinical global improvement compared with placebo in a 6-week trial. Controlled studies have also found the tricyclic antidepressants (TCAs) desipramine¹⁸ and imipramine¹⁹ to be superior to placebo in the similarly defined conditions of nonpurging bulimia nervosa and binge eating with obesity, respectively.

Several lines of evidence suggested to us that the novel antidepressant venlafaxine might be particularly effective in treating binge-eating disorder, including binge eating associated with obesity. First, venlafaxine blocks the reuptake of norepinephrine as well as serotonin.²⁰ Thus, venlafaxine shares the serotonin reuptake blocking properties of SSRIs, the norepinephrine reuptake blocking properties of TCAs and bupropion²¹ (which have been shown to be superior to placebo in reducing binge eating associated with bulimia nervosa^{16,22}), and the serotonin-norepinephrine reuptake blocking properties of the anti-obesity agent sibutramine.^{23,24} Second, unlike TCAs but like bupropion,²⁵ venlafaxine²⁶ has been associated with weight loss in patients with depression. In addition, bupropion has been shown to be superior to placebo in reducing weight associated with obesity in a controlled study.²⁷ Third, a recent meta-analysis of studies comparing venlafaxine with SSRIs concluded that venlafaxine was more efficacious than SSRIs in the treatment of major depression, possibly due to the drug's dual reuptake of serotonin and norepinephrine.²⁸ Data from epidemiologic, phenomenologic, comorbidity, family history, and treatment response studies suggest that binge eating and major depression may be related.^{2,4,8,11-19,29-31} Twin data further suggest that binge eating and depression may share common genetic factors.³² It could, therefore, be hypothesized that its dual mechanism of action might also make venlafaxine more efficacious than SSRIs in the treatment of binge-eating disorder.

On the basis of this rationale and our initial clinical observations of several patients with binge-eating disorder resistant to SSRIs who responded favorably to venlafaxine, we systematically reviewed the charts of the first 35 consecutive outpatients with DSM-IV binge-eating disorder¹ who received clinical treatment with venlafaxine at our weight management program.

METHOD

The medical charts of the first 35 consecutive outpatients with binge-eating disorder by DSM-IV criteria¹ who were treated with venlafaxine at the University of Cincinnati Physicians Weight Management Program, Cincinnati, Ohio, were retrospectively identified. Chart notations confirmed that all patients had provided written consent to receive venlafaxine.

Participation at our weight management program included a thorough psychiatric evaluation (Structured Clinical Interview for DSM-IV³³ and review of psychiatric, medical, and dietary treatment histories) and appropriate psychopharmacologic treatment of identified eating, mood, and other psychiatric disorders that might be contributing to overeating, physical inactivity, or both. Behavioral dietary counseling with a registered dietitian was strongly encouraged but not mandatory. Behavioral

dietary counseling focused on healthy eating, exercise, and other lifestyle changes; it did not include the use of diets or caloric restriction. Patients with binge-eating behavior were instructed to maintain binge diaries. Formal cognitive-behavioral or insight-oriented therapy was not provided. Patients who required such psychotherapy were given referrals to local licensed clinicians well known to the authors.

Response and side effects to medication were evaluated in face-to-face interviews via retrospective recall at approximately monthly appointments. Response of binge-eating symptoms was routinely assessed by review of binge diaries and patients' report of their weekly binge frequency. The number of binges the patient reported in the week prior to the clinic visit was regularly recorded in the progress note. The Clinical Global Impressions-Severity of Illness (CGI-S) scale³⁴ was used to assess the patient's binge-eating behavior as well as any comorbid mood, anxiety, and other disorders. The CGI-S scale is a 7-point scale on which 1 = normal, 2 = borderline ill, 3 = mildly ill, 4 = moderately ill, 5 = markedly ill, 6 = severely ill, and 7 = among the most extremely ill patients.

We systematically reviewed the charts of the 35 patients receiving venlafaxine for binge-eating disorder and recorded their demographic, psychiatric, medical, and treatment information. Specifically, patients' responses to venlafaxine, BMIs (weight in kilograms divided by height in meters squared), weights, waist circumferences, and vital signs were recorded at each clinic visit, as well as whether they had received behavioral dietary counseling from our registered dietician, were referred for formal psychotherapy with a licensed provider, or both. Patients' response to venlafaxine was assessed by recording the estimated weekly binge frequency (the number of binges the patient reported in the week prior to the clinic visit) and the CGI-S scale for binge eating. In addition, each patient was assigned a response category at each visit based on the change in weekly binge frequency at that visit compared with the visit at which venlafaxine was begun. For response categories, "no response" was a 0% to 24% decrease in weekly binge-eating episodes; "mild" was a 25% to 49% decrease in binge-eating episodes; "moderate" was a 50% to 74% decrease in weekly binge-eating episodes; "marked" was a 75% to 99% decrease in binge-eating episodes; and "remission" was complete cessation of binge-eating episodes.

Three statistical analyses were performed. The main analysis examined the effect of venlafaxine monotherapy or adjunctive therapy in binge-eating disorder and included the 33 women who received venlafaxine alone or in combination with other antidepressants while actively binge eating. The second examined venlafaxine's effect on obesity associated with binge-eating disorder, no matter the current status of binge eating, and included all 35 women, because prospective studies have shown that

Table 1. Baseline Characteristics of 35 Women With Binge-Eating Disorder and Overweight or Obesity Treated With Venlafaxine^a

Feature	Mean \pm SD	Range
Age, y	45.9 \pm 9.3	28–68
Binges/wk ^b	4.2 \pm 2.7	0–10
CGI-S (binge eating) ^c	4.5 \pm 1.4	1–7
CGI-S (mood)	3.9 \pm 1.3	1–7
BMI (kg/m ²) ^d	39.0 \pm 8.0	29.0–61.0
Weight (lb) ^e	230.0 \pm 51.6	163.0–378.2
Waist circumference (in)	40.5 \pm 5.7	32.8–57.0
Blood pressure (mm Hg)		
Systolic	127.1 \pm 16.6	102–170
Diastolic	81.0 \pm 8.1	68–100
Pulse (bpm)	75.1 \pm 12.1	52–100

^aAbbreviations: BMI = body mass index, CGI-S = Clinical Global Impressions-Severity of Illness scale.

^bMean \pm SD (range) binges/week = 4.5 \pm 2.5 (1–10) in 33 patients with active binge eating.

^cMean \pm SD (range) CGI-S (binge eating) = 4.9 \pm 0.8 (3–7) in 33 patients with active binge eating.

^dMean \pm SD (range) BMI = 39.1 \pm 8.1 (29–67) in 33 patients with active binge eating.

^eMean \pm SD (range) weight = 231.8 \pm 51.8 lb (163.2–378.2 lb) in 33 patients with active binge eating.

binge eating is associated with the development of obesity even when persons report cessation of binge eating.¹¹ The third focused on venlafaxine monotherapy in binge-eating disorder and included the 29 women who received venlafaxine alone while actively binge eating.

For the statistical analyses, the difference between the baseline observation and the last available observation on each endpoint was computed for each patient. A *t* test of the hypothesis that the mean change was equal to 0 was carried out for each endpoint at $\alpha = .05$. In addition, for the main analysis, correlations were performed between both venlafaxine treatment dose and duration and change in weekly binge frequency, change in weight, and change in BMI. Correlations were also performed among changes in psychiatric variables (weekly binge frequency, CGI-S scale scores for binge eating and mood disorder) and changes in both anthropometric variables and vital signs (weight, BMI, waist circumference, systolic and diastolic blood pressure, and pulse).

RESULTS

The clinical characteristics of the 35 patients are summarized in Table 1. All 35 patients were female, and 33 patients were actively binge eating at the time venlafaxine therapy was begun. Thirty-two patients (91%) were obese (BMI \geq 30.0), and 3 (9%) were overweight but not obese (BMI 25.0–29.9). All patients had at least 1 lifetime comorbid Axis I disorder in addition to binge-eating disorder; 30 (86%) had a mood disorder (all depressive disorders), 24 (69%) had an anxiety disorder, 8 (23%) had a past substance use disorder, and 4 (11%) had past bulimia nervosa. Twelve patients (34%) had a diagnosis of hypertension and 11 (31%) were receiving antihypertensive

medication. Thirty-two patients (91%) received behavioral dietary counseling, 1 (3%) received formal psychotherapy, and 1 (3%) received both modalities within 3 months of initiation of venlafaxine.

Venlafaxine was given as monotherapy to 29 patients (83%) and adjunctively to the remaining 6 patients (17%) for a median of 120 days (range, 28–300 days). The extended release formulation was used in all patients. Venlafaxine was usually begun at 37.5 mg in the morning and subsequently increased by 37.5 to 75 mg/week according to the patient's response and side effects to a maximum dose of 300 mg/day, usually given once a day. The mean \pm SD venlafaxine treatment dose was 222 \pm 63 mg/day (range, 75–300 mg/day). In the patients receiving venlafaxine adjunctively, the drug was added to amitriptyline (N = 1), bupropion (N = 2), paroxetine (N = 2), and sertraline (N = 1).

In the 33 patients receiving venlafaxine alone or in combination while actively binge eating, weekly binge frequency, CGI-S scale scores for binge eating and mood (depressive) disorder, weight, BMI, waist circumference, and diastolic blood pressure all showed statistically significant decreases over time (Table 2). These variables also showed statistically significant decreases over time in the entire group of 35 patients with overweight or obesity and in the 29 patients receiving venlafaxine monotherapy while actively binge eating (see Table 2).

Regarding categorical response, 29 (88%) of the 33 patients who were actively binge eating displayed a moderate or better response of binge eating to the addition of venlafaxine. The other 4 patients displayed a mild (N = 1) or no response (N = 3). Of the 29 patients who displayed a moderate or better response in their binge eating, 13 (45%) lost 5% or more of their baseline weight; 6 (21%) lost 10% or more of their baseline weight. Fifteen (43%) of the 35 patients showed a clinically significant loss of weight, defined as losing 5% or more of their baseline weight; 7 (20%) lost 10% or more of their baseline weight.

Venlafaxine treatment dose correlated with change in CGI-S scale scores for binge eating ($r = -0.40$; $p = .02$), change in weight ($r = -0.37$; $p = .04$), and change in BMI ($r = -0.41$; $p = .02$), but not with change in binge frequency ($r = -0.31$; $p = .08$). Duration of treatment with venlafaxine did not correlate with any of these variables. Moreover, there were no correlations between change in weekly binge frequency and any anthropometric measures (weight, BMI, and waist circumference) or vital signs (systolic and diastolic blood pressure and pulse). However, change in CGI-S scale scores for binge eating correlated with change in weight ($r = 0.63$; $p < .0001$) and change in BMI ($r = 0.62$; $p < .0001$), and change in CGI-S scale scores for mood (depressive) disorder correlated with change in waist circumference ($r = 0.42$; $p < .03$).

Table 2. Baseline-to-Endpoint Mean Change and 95% Confidence Limits in Clinical Variables in Women With Binge-Eating Disorder and Overweight or Obesity Treated With Venlafaxine^a

Variable	Active BED (venlafaxine alone or in combination) (N = 33)		Active and/or Inactive BED (venlafaxine alone or in combination) (N = 35)		Active BED (venlafaxine monotherapy) (N = 29)	
	Change (range)	p Value	Change (range)	p Value	Change (range)	p Value
Binges/wk	-3.65 (-4.72, -2.59)	< .0001	-3.44 (-4.49, -2.40) ^c	< .0001	-3.88 (-5.07, -2.69)	< .0001
CGI-S (binge eating)	-2.00 (-2.59, -1.41)	< .0001	-1.89 (-2.47, -1.30) ^d	< .0001	-2.07 (-2.74, -1.40)	< .0001
CGI-S (mood)	-1.59 (-2.06, -1.12) ^b	< .0001	-1.55 (-1.99, -1.11) ^e	< .0001	-1.63 (-2.14, -1.11) ^f	< .0001
Weight (lb)	-10.90 (-15.18, -6.62)	< .0001	-11.00 (-15.12, -6.89)	< .0001	-10.90 (-15.62, -6.19)	< .0001
BMI	-2.00 (-2.75, -1.25)	< .0001	-2.00 (-2.73, -1.27)	< .0001	-2.00 (-2.83, -1.17)	< .0001
Waist circumference (in)	-1.95 (-2.76, -1.15)	< .0001	-2.01 (-2.80, -1.23)	< .0001	-1.85 (-2.64, -1.06)	< .0001
Blood pressure (mm Hg)						
Systolic	-4.09 (-10.11, 1.92)	.1755	-3.29 (-9.05, 2.48)	.26	-3.55 (-10.29, 3.19)	.290
Diastolic	-4.94 (-8.96, -0.92)	.0176	-5.23 (-9.16, -1.30)	.01	-5.00 (-9.51, -0.49)	.031
Pulse (bpm)	4.24 (0.17, 8.32)	.0419	4.86 (0.86, 8.86)	.02	4.55 (0.04, 9.06)	.048

^aAbbreviations: BED = binge-eating disorder, BMI = body mass index, CGI-S = Clinical Global Impressions-Severity of Illness scale.

^bPerformed only for those 27 patients with comorbid mood (depressive) disorders.

^cIncludes 2 patients reporting no bingeing at baseline.

^dIncludes 2 patients with CGI-S (binge eating) = 0 at baseline.

^ePerformed only for those 29 patients with comorbid mood (depressive) disorders.

^fPerformed only for those 24 patients with comorbid mood (depressive) disorders.

In general, venlafaxine was well tolerated. The most commonly reported side effects among the 35 patients were dry mouth (23%, N = 8), sexual dysfunction (14%, N = 5), insomnia (14%, N = 5), and nausea (11%, N = 4). Sixteen patients (46%) showed blood pressure changes (defined as an increase or decrease by a minimum of 10 points in systolic or diastolic blood pressure for a minimum of 3 consecutive visits); 6 showed a sustained increase, 9 showed a sustained decrease, and 1 showed both (an initial increase followed by a sustained decrease; this patient was classified as showing a sustained decrease, see below). One patient whose blood pressure initially increased normalized by her seventh clinic visit; the increase persisted in the other 5 patients. The median baseline blood pressure in the 6 patients displaying a sustained increase was 110 (range, 102 to 135)/72 (range, 68 to 78) mm Hg; the median change was +11.5 (range, -5 to +30) for systolic pressure and +4 (range, +2 to +18) for diastolic pressure. The median baseline blood pressure in the 10 patients displaying a sustained decrease was 140 (range, 110 to 160)/88 (range, 78 to 100) mm Hg; the median change was -21 (range, -38 to -10) for systolic and -13.5 (range, -28 to 0) for diastolic blood pressure. None of the blood pressure increases were thought to be clinically significant and many of the decreases were thought to be beneficial. There was also a small but statistically significant increase in pulse over time ($p = .0419$) (see Table 2). No patient discontinued the drug.

DISCUSSION

In this open, retrospective review, patients with binge-eating disorder and overweight or obesity receiving venlafaxine at a weight management program displayed improvement in both binge eating and overweight. Over

time, venlafaxine treatment was associated with statistically significant reductions of binge-eating frequency, CGI-S scale binge-eating and mood disorder scores, weight, BMI, waist circumference, and diastolic blood pressure.

Specifically, 29 (88%) of 33 actively binge-eating patients reported a 50% or greater reduction in their binge eating, and 15 (43%) of the entire group of 35 patients showed a 5% or greater reduction in baseline weight. Of the 33 patients who were actively binge eating, 13 (39%) showed clinically significant improvement in both binge eating and weight (a 50% or greater reduction in binge eating and a 5% or greater reduction in baseline weight).

Venlafaxine was generally well tolerated; no patient discontinued the drug. Although 6 patients developed sustained blood pressure elevations, none of the elevations were considered clinically significant.

These findings are limited by several methodological flaws. Most importantly, venlafaxine treatment was naturalistic and open label, and thus nonrandomized, unblinded, and uncontrolled. The possibility that the observed favorable response of binge eating, overweight, or both to venlafaxine was in fact due to placebo response, clinician or patient bias, or spontaneous remission cannot be excluded. Second, most patients were receiving other treatments. Specifically, 32 patients received behavioral dietary counseling, 1 received formal psychotherapy, and 1 received both. Also, in 6 patients, venlafaxine was added to ongoing antidepressant therapy. Thus, the reduction in binge eating, overweight, or both could be attributed to the other treatments patients were receiving. Of note, although available studies suggest that behavioral dietary therapy, cognitive-behavioral therapy, interpersonal therapy, and TCAs are effective in reducing binge eating in obese binge eaters, their effectiveness in reduc-

ing overweight in this patient group is less clear.³⁵⁻³⁸ A third limitation of our findings is that our patients received venlafaxine for a median of only 120 days. It is, therefore, unknown if our findings would generalize to longer durations of treatment. A fourth limitation is that the majority of our patients had psychiatric disorders other than binge-eating disorder, including disorders known to respond to venlafaxine. These included major depressive disorder and various anxiety disorders. Thus, it is unknown whether venlafaxine would reduce binge eating or overweight in the absence of these comorbid disorders.

These findings must, therefore, be regarded as highly preliminary. Nonetheless, the response in both binge eating and overweight observed in 13 (39%) of 33 patients with active binge-eating disorder and obesity suggests that venlafaxine may have utility for at least a subset of persons with binge-eating disorder. Controlled trials of venlafaxine in binge-eating disorder appear warranted.

Drug names: bupropion (Wellbutrin and others), desipramine (Norpramin and others), fluvoxamine (Luvox and others), paroxetine (Paxil), sertraline (Zoloft), sibutramine (Meridia), venlafaxine (Effexor).

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