Predictors of Binge Eating in Restrictive Anorexia Nervosa Patients in Italy

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Abstract: The aim of the present study was to investigate the predictive factors for the development of binge eating in restricting anorexia nervosa patients who underwent an outpatient treatment. The sample was a group of 168 patients with restricting anorexia nervosa, consecutively admitted to an outpatient unit. All the patients underwent a routine baseline assessment. Information regarding binge eating onset and duration of treatment was prospectively recorded in clinical records. Twenty-three patients developed binge episodes during outpatient treatment. High scores on the Eating Disorders Inventory body dissatisfaction subscale and early age at menarche significantly predict the risk of developing binge eating. High levels of ineffectiveness, as measured by the Eating Disorders Inventory, and early age at menarche are significant predictors of a shortened time to develop binge eating. The identification of subjects at risk for developing binge eating should be considered an important step for a successful treatment of restricting anorexic patients.

Key Words: Anorexia nervosa, binge eating, menarche, weight recovery.

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Although it is widely accepted within the research and clinical literature that anorexia nervosa (AN) and bulimia nervosa (BN) are two different conditions defined by different diagnostic criteria, there are too many similarities between them to consider these disorders as two completely independent situations (Fairburn and Harrison, 2003). AN and BN seem to share several psychological and environmental risk factors (Stice, 2002) as well as some genetic liability (Strober et al., 2000). Furthermore, it has been estimated that a high percentage of eating disordered subjects tend to migrate between the diagnostic categories (Fairburn and Harrison, 2003; Favaro et al., 2003; Tozzi et al., 2005). However, few studies have investigated the predictive factors for diagnostic crossover in eating disorders (Bulik et al., 1997; Strober et al., 1997; Tozzi et al., 2005).

Long-term outcome studies of AN patients indicate that up to 64% of subjects develop binge eating in their lifetime (Bulik et al., 1997; Strober et al., 1997; Tozzi et al., 2005). In addition, the diagnostic criteria for lifetime AN are met in 10% to 54% of BN cases (Favaro et al., 2003; Sullivan et al., 1996). The most vulnerable period seems to be the first 5 years from the onset of AN (Bulik et al., 1997; Strober et al., 1997).

The present study aims at investigating the predictors of the development of binge eating in restricting AN patients who are following an outpatient cognitive-behavioral treatment of their eating disorder.

METHODS

All subjects fulfilling the DSM-IV diagnostic criteria for restricting AN referred to the Padova Eating Disorders Unit in the period June 1993 to June 2002 were considered eligible for the study. Only subjects who underwent an outpatient treatment of a period of at least 3 months were included. Criteria of exclusion were presence or history of bingeing or purging episodes and presence of a severe medical or neurological comorbidity. Informed consent was obtained from all subjects. The protocol was in accord with the Helsinki Declaration of 1975 as revised in 1983.

The final sample consisted of 168 patients (163 females and five males) with a mean age at presentation of 20.6 years (range 12–36; $SD = 4.6$). At presentation, each patient underwent a structured diagnostic interview (Eating Disorders section of the Structured Clinical Interview for DSM-IV; First et al., 1995) and a semistructured interview to gather clinical data. The Hopkins Symptom Checklist (SCL-90; Derogatis et al., 1976), the Eating Disorders Inventory (EDI; Garner et al., 1984), and the Tridimensional Personality Questionnaire (Cloninger, 1987) were administered as part of the routine initial assessment. SCL-90 was completed by 161 (96%), EDI by 160 (95%), and Tridimensional Personality Questionnaire by 110 subjects (65%).

Clinical data about the onset of binge eating and general outcome were prospectively recorded from presentation to the end of treatment. The mean time of treatment was 16.8 months (range 3–66; $SD = 12.9$). Binge eating patients were defined as all the subjects who presented at least four episodes of objective binge eating in a consecutive period of 2 weeks.

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Statistics

Groups were compared by means of two-tailed *t* test and χ^2 (with Yates' continuity correction for 2×2 tables). Given the presence of multiple comparisons, only *p* values less than 0.005 were considered significant. Stepwise multiple logistic regression analysis was employed to find the predictors of binge eating and multivariate Cox regression modeling to test the effects of predictor variables on time to develop binge eating.

RESULTS

Twenty-three (14%) subjects developed binge eating at some time during treatment. On average, ANR subjects developed binge eating after 6.7 (*SD* = 6.4) months of treatment at a BMI of 17.3 (*SD* = 1.5)—that is, after having regained 9.8% (*SD* = 12.4) of their initial body weight. The BMI at the onset of binge episodes was correlated with the BMI at the baseline (Spearman $\rho = 0.52$; $p < 0.02$).

Table 1 shows the differences between subjects who developed binge eating and those who did not as regards some of the considered variables. The binge eating group did not differ from the other subjects as regards age, duration of illness, age of onset, BMI at presentation, number of previous treatments, hours per week of excessive exercise, history of childhood sexual/physical abuse, substance use and/or alcohol abuse, and temperamental variables (Table 1).

The presence of stronger body dissatisfaction at baseline (measured by the EDI) and early age at menarche were the predictors of binge eating in a stepwise logistic regression analysis (EDI body dissatisfaction: B = 0.11, Wald = 10.14, $p < 0.002$, Exp(B) = 1.11; age at menarche: B = −0.26, Wald = 3.70, $p = 0.054$, Exp(B) = 0.77). The variables yielded a model χ^2 value of 14.74 ($df = 2$; $p < 0.002$). Two variables made a significant contribution to the prediction of time to develop binge eating after the starting of the treatment: the final model resulting from the Cox regression model indicated that high levels of ineffectiveness, as measured by the EDI, and early age at menarche are significant predictors of a shortened time to develop binge eating (EDI ineffectiveness: B = 0.07, Wald = 7.37, $p < 0.01$, Exp(B) = 1.07; age at menarche: B = −0.25, Wald = 7.32, $p < 0.01$, Exp(B) = 0.78). The variables yielded a model χ^2 value of 13.57 ($df = 2$; $p < 0.001$).

Since in our sample we included all the subjects who underwent at least 3 months of outpatient treatment and the mean time at onset of binge eating was 6.7 months, we repeated all the analyses only for subjects who remained in treatment of at least 12 months. In this group (94 subjects),

TABLE 1. Differences Between the Binge Eating Patients and Those Who Did Not Develop Binge Eating

	Binge Eating AN (*N* = 23) Mean (*SD*)	Nonbinge Eating AN (*N* = 145) Mean (*SD*)	*t*	*p*
Age	20.2 (4.0)	20.8 (4.8)	0.54	0.589
Duration of illness	22.6 (20.5)	28.7 (34.8)	0.82	0.414
Age at onset of AN	17.5 (4.0)	17.4 (3.9)	0.09	0.926
BMI at intake	15.9 (1.6)	15.3 (1.6)	1.77	0.078
Premorbid BMI	23.1 (4.2)	20.4 (2.5)	3.81	0.001
BMI at amenorrhea	19.1 (1.9)	17.4 (1.9)	2.81	0.007
Age at menarche	12.0 (3.0)	12.9 (1.3)	2.21	0.028
EDI drive for thinness	12.8 (7.4)	7.8 (6.9)	3.23	0.002
EDI bulimia	3.0 (3.2)	1.5 (1.9)	2.01	0.046
EDI body dissatisfaction	14.8 (7.7)	9.8 (6.4)	3.05	0.003
EDI ineffectiveness	11.7 (8.6)	7.0 (6.4)	3.35	0.001
EDI perfectionism	6.0 (4.3)	4.1 (3.4)	3.12	0.002
EDI interpersonal distrust	7.5 (5.2)	6.2 (4.5)	1.84	0.068
EDI interoceptive awareness	9.6 (7.0)	6.8 (6.0)	2.32	0.022
EDI maturity fears	9.4 (5.5)	7.4 (4.8)	1.28	0.202
SCL total score	1.3 (0.8)	1.0 (0.6)	1.80	0.074
SCL somatization	1.0 (0.7)	0.8 (0.7)	0.94	0.351
SCL obsessive-compulsive	1.3 (0.9)	1.2 (0.8)	0.98	0.329
SCL depression	1.7 (1.1)	1.3 (0.8)	1.95	0.053
SCL anxiety	1.4 (1.0)	1.1 (0.8)	1.36	0.177
SCL hostility	1.3 (0.9)	0.9 (0.7)	2.28	0.024
TPQ novelty seeking	14.3 (4.7)	13.6 (4.8)	0.53	0.595
TPQ harm avoidance	21.8 (3.9)	20.5 (6.4)	0.74	0.463
TPQ reward dependence	13.9 (3.5)	14.6 (4.1)	0.63	0.528
TPQ persistence	6.5 (1.2)	6.0 (1.7)	1.04	0.299

the rate of onset of episodes of objective binge eating was 22.3%. All the statistical significant differences reported in Table 1, and the predictors found by the logistic regression and Cox regression analyses were confirmed.

DISCUSSION

The investigation of the predictors of diagnostic crossover in the field of eating disorders is important for multiple reasons. First of all, the occurrence of binge eating should be considered an important complication in the course of restricting AN, because it has a negative prognostic impact (Steinhausen, 2002) and seems to delay the time to full recovery (Strober et al., 1997). In the only available prospective study of the predictors of binge eating, the sample was formed by adolescent restricting AN subjects discharged from an intensive inpatient treatment program. Our study is the first to investigate the predictors of the onset of binge eating during the course of a cognitive-behavioral treatment conducted, from the beginning, in an outpatient setting. Since outpatient treatment can be considered the first-choice approach in AN cases without medical complications (Wilson and Shafran, 2005), this type of design is the most appropriate to give useful clinical indicators about which patients are more at risk and in which phase of their treatment. This type of knowledge is fundamental to plan specific preventive measures to avoid the onset of bulimic symptoms. Furthermore, from a theoretical point of view, it is important to gain further knowledge about the significance of the diagnostic instability for the definition of the phenotype of eating disorders. Lifetime crossover from AN to BN and vice versa appears to occur in about half the subjects with a clinical eating disorder (Tozzi et al., 2005). Although it is important to use a uniform definition of the eating disorder phenotype to find susceptibility genes (Bulik et al., 2003a; Grice et al., 2002), diagnostic crossover might be considered an essential component of the phenotype of eating disorders, whose genetic and environmental risk factors should be investigated in future studies.

In the present study, we have found a rate of diagnostic crossover from restricting AN to BN lower than that found in the prospective study of Strober et al. (1997) and those found in retrospective studies (Bulik et al., 1997; Tozzi et al., 2005). The main reason for this is probably the number of truncated observations in our study. Another possible explanation is the exclusion from our sample of all patients who reported recurrent or sporadic bulimic/purging symptoms in the past.

Restricting AN patients who develop episodes of objective binge eating are characterized by two main factors: an earlier age at menarche, and the presence of more severe body dissatisfaction. Furthermore, high levels of ineffectiveness and an early age at menarche seem to indicate that binge eating can occur in the very first phases of treatment. Early age at menarche has been implicated as a risk factor for the development of bulimia nervosa in previous studies (Fairburn et al., 1997). Fairburn et al. (1997) have observed that early menarche can be considered a risk factor for dieting because it involves early exposure to the changes in body shape due to puberty. It is also possible that early menarche is associated with the presence of an excess of fat mass that further increases the risk of dieting behavior. In our sample, binge eating patients reported amenorrhea at a higher weight and had a higher premorbid body mass index. It is possible that, in subjects with AN, the risk of developing bulimic episodes is linked to factors which have an impact on the regulation of body weight and puberty timing/menstrual regulation. This hypothesis would be supported by the observation that patients of the binge eating/purging type AN have, on average, an higher current and lifetime body mass index than AN subjects of the restricting type (Hebebrand and Remschmidt, 1995; Vervaet et al., 2004). Other, more psychological, factors seem to have a role in the risk of developing binge eating in our sample. AN patients with a more intense body image disturbance who are unable to cope with their emotions and sensations are to be considered more at risk for developing binge eating. Other personality or psychopathological characteristics, such as anxiety, depression, or novelty seeking, did not seem to have a specific role in increasing the risk of binge eating in our sample of AN subjects.

Although we have used a prospective design, our findings cannot be compared with those of outcome studies, where the time of observation is not limited to the duration of treatment. Moreover, generalization of our findings is limited by the variability of time of observation. However, the naturalistic approach of the present study can give therapists some useful indicators for an early identification of subjects at risk for developing binge eating during an outpatient treatment. Restricting AN subjects with an early age at menarche and high scores at the EDI seemed to be more at risk for this complication and, usually, they appeared to begin binge eating after a partial weight restoration. The effectiveness of psychological and psychopharmacological treatments (Santonastaso et al., 2001) in the prevention or early treatment of this complication should be evaluated in future studies, since the onset of binge eating has a negative prognostic value and appears to require a more intensive therapeutic effort.

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