ORIGINAL ARTICLE

Individualized treatment of eating disorders

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Abstract

OBJECTIVE: The goal of this study is to determine how a comprehensive approach comprising a combination of a particular psychotherapeutic programme and antidepressant treatment influences the signs and symptoms of eating disorders.
METHODS: Inpatients entering a particular 6-week programme were assessed using body mass index (BMI), Eating Disorder Inventory (EDI), Eating Attitudes Test (EAT-26) and psychological symptoms using Symptom Checklist (SCL-90).
RESULTS: In the whole group (n=84) the mean BMI remained stable (17.9, 17.8). There was a significant effect of TIME in the repeated measures ANOVA with most of dependent variables. The "TREATMENT" had no effect (antidepressants, no antidepressants) and there was no significant interaction between "TREATMENT" and "TIME". Post-hoc analysis revealed significant differences between baseline and end-point values of all but two EDI, all EAT items and some SCL dimensions in the antidepressant-treated group.
CONCLUSIONS: The comprehensive and individualized approach is able to achieve weight stabilization, and improvement in attitudes, pathological eating behaviour and psychopathology.

Abbreviations:

AN	- anorexia nervosa
BN	- bulimia nervosa
AD	- antidepressants
SSRI	- specific serotonin reuptake inhibitors
ICD-10	- The tenth Revision of the International Classification of Diseases
BMI	- body mass index
EDI	- Eating Disorder Inventory
EAT-26	- Eating Attitudes Test
SCL-90	- Symptom Checklist
GSI	- Global Severity Index

INTRODUCTION

Patients with eating disorders display a broad range of symptoms that frequently occur along a continuum between those of anorexia (AN) and bulimia nervosa (BN). The care of patients with eating disorders involves a comprehensive array of approaches. Psychiatric management should form the basis of the treatment for patients with eating disorder. The treatment options generally include: nutritional rehabilitation – refeeding, psychosocial interventions, medications (APA 2000; Papezova *et al.* 2006). Individuals with eating disorders, especially anorexia nervosa, are inherently difficult to treat, even in an inpatient setting.

The aims of the AN treatment are to restore weight, treat physical complications, enhance patient motivations to cooperate in the restoration of healthy eating patterns and to participate in treatment, correct core maladaptive thoughts, attitudes, and feelings related to the eating disorder, treat associated psychiatric conditions and prevent relapse. Nutritional rehabilitation should be established for all patients who are significantly underweight. Once weight gain has started, formal psychotherapy may be very helpful. There is no clear evidence that any specific form of psychotherapy is superior for all patients. The role of antidepressants (AD) is usually best assessed following weight gain, when the psychological effects of malnutrition are resolving. ADs should be considered for the prevention of relapse in weight – restored patients or to treat associated features such as depression or obsessive-compulsive problems (APA 2000; Papezova *et al.* 2006).

ADs are effective as one component of an initial treatment programme for most patients with BN. Specific serotonin reuptake inhibitors (SSRI) are currently considered to be the safest AD and may be especially helpful for patients with significant symptoms of depression, anxiety, obsession or certain impulsive disorder symptoms or for those patients who have had a suboptimal response to previous attempts at appropriate psychosocial therapy. Emerging evidence has shown that a combination of psychotherapeutic interventions and medication should be considered when initiating treatment of patients with BN (Mitchell *et al.* 2007).

There are some other promising treatment strategies which could be acceptable by patients suffering from eating disorders, like bright light therapy. Besides its antidepressant effect light therapy may help to restore the irregular circadian rhytmicity observed in some subjects (Yamamotova *et al.* 2008).

Our department specializes in the comprehensive treatment of eating disorder. We are able to provide care for patients from children to adults and to deal with the whole spectrum of eating disorder severities – form intensive psychiatric care to outpatient treatment. The inpatient module usually lasts from 6 to max. 8 weeks. There is very close cooperation with the intensive care unit, children's ward, and dietary assistants.

The primary objectives of this prospective naturalistic study are:

1. to determine in what way the comprehensive approach comprising a combination of a particular psychotherapeutic programme and antidepressant treatment influence the signs and symptoms of eating disorders;
2. to determine the effect of an antidepressant add-on on weight, the symptoms of eating disorders and general psychopathology;
3. to determine the differential effect of SSRI vs. mirtazapine on signs and symptoms of eating disorders.

METHODS

Participants

Patients suffering from eating disorders (according to ICD 10) consecutively hospitalized (waiting list average 3 months) between January 2002 and December 2006 in the Eating Disorders Unit, who entered a particular programme and were able to undergo the programme (severely malnourished subjects were excluded) were included. The diagnosis was confirmed by the consensus of two psychiatrists in two separate interviews.

Treatment

The patients entered a special psychotherapeutic programme. This 6-week programme for eating disorders is based mainly on work in a closed psychotherapeutic group. Emphasis is put on re-establishment of healthy eating habits and weight stabilization. Psychotherapeutic work is centred on enhancement of self perception, self regulation, and resolving underlying psychological and interpersonal conflicts. Groups are psychodynamic, semi-structured, using art therapeutic techniques and imaginations (Guided Affective Imagery). Patients were treated with ADs according to clinical need; in most cases for associated depressive or anxiety symptoms. Two groups of ADs were used: SSRIs and dual AD mirtazapine. The daily doses have been individualized.

Assessment

Assessments include anthropometric variables (height, weight and BMI), specific measures for eating disorders – EDI, EAT-26 and a specific measure for psychological symptoms – SCL-90 at the beginning and at the end of the programme.

The EDI is a 64-item, self-report questionnaire, designed to provide information on eight separate dimensions of cognitive and behavioural aspects of anorexia nervosa and bulimia. The EDI consists of 8 subscale scores: DRIVE FOR THINNESS, BULIMIA, BODY DISSATISFACTION, INEFFECTIVENESS, PERFECTIONISM, INTERPERSONAL DISTRUST, INTEROCEPTIVE AWARENESS, and MATURITY FEARS. Completion takes 15–25 minutes (Garner *et al.*2006). EAT is a brief (26 item), standardized, self-

report screening test of symptoms, and concerns eating disorder characteristics. Completion time is 5–10 minutes (Garner *et al.* 1982). The EAT-26 items form three subscales, DIETING, BULIMIA AND FOOD PREOCCUPATION and ORAL CONTROL. General psychopathology was assessed using the SCL-90-R. This is a 90-item self-report system inventory developed in the 1980s by Derogatis and designed to reflect the psychological symptom patterns of community, medical and psychiatric respondents. The SCL-90-R (Derogatis *et al.* 1973) is a simple questionnaire that has recently been validated in a number of clinical conditions related papers (Wallis *et al.* 1998). Each of the items is rated on a five-point scale of distress (0–4) ranging from not at all to extremely. The nine primary symptom dimensions are: SOMATIZATION, OBSESSIVE-COMPULSIVE, INTERPERSONAL SENSITIVITY, DEPRESSION, ANXIETY, HOSTILITY, PHOBIC ANXIETY, PARANOID IDEATION, and PSYCHOTICISM. There are also three global indices: global severity index (GSI), positive symptom distress and positive symptom total. High test-retest and internal consistency have been demonstrated, and there do not appear to be any problems with the practical effect. Normally the test can be completed in about 10–15 minutes. For evaluation of treatment effect on psychopathology the GSI and raw scores for dimensions were used. Raw scores are calculated by dividing the sum of scores for a dimension by the number of items in the dimension. The GSI is then computed by first summing the scores of the nine dimensions and the additional items, then dividing by the total number of responses (i.e., 90, unless some questions were unanswered).

Statistics

The analysis was based on descriptive statistics; the distribution of categorical variables was tested using the chi-quadrate test, and the distribution of continuous variables was analysed using parametric methods (two-sample t-test) with the level of statistical significance set to $p<0.05$. The effect of antidepressant add-on was tested using repeated measures ANOVA with parameters "TREATMENT" (AD, NO AD) and "TIME" (baseline and end-point). For the analysis only observed cases have been used. The differential effect of SSRI and mirtazapine was tested using repeated measures ANOVA with parameters "AD GROUP" (SSRI, mirtazapine, no antidepressants – NO AD) and "TIME" (baseline and end-point). Post-hoc Fisher´s LSD test was used to analyze individual group differences. To deal with the multiple comparisons problem the Bonferroni correction was used with the level of statistical significance set to $p<0.001$ analysis.

Tab. 1. Sample characteristics (n=84).

	Mean values (SD)
Age	22.6 (±4)
Age at the onset of the illness	15.6 (±3)
High (m)	1.67 (±0.6)
Weight on admission (kg)	50.1 (11.0)
Weight at discharge (after the programme)	50.2 (9.9)
BMI on admission - anorexia - bulimia	17.9 (3.3) 15.3 (2.1)*** 20.1 (2.5)
BMI at discharge - anorexia - bulimia	17.8 (3.6) 15.7 (2.0)*** 19.6 (3.9)

*** $p<0.001$…pts suffering from anorexia had signif. lower BMI than pts suffering from bulimia.

RESULTS

Whole sample characteristics

84 patients (80 females) suffering from AN (both restrictive and purgative type) and BN were included. 41 patients were suffering from BN, 37 from AN and 6 with atypical forms of both BN and AN. The mean age was 22.6 (±4) years. The mean age of illness onset was 15.6 (±3) years. In the whole group the mean weight and BMI on admission and at discharge remained the same (50.1 and 50.2 respectively; 17.9 and 17.8 respectively). Patients with AN had significantly lower weight and BMI both on admission and at discharge (see Table 1).

Eight patients (9.5%) did not complete the comprehensive treatment programme. Patients who dropped out had significantly higher scores in the EDI items "BULIMIA" (11.1 vs. 6.2, t=2.1, $p=0.04$), "PERFECTIONISM" (8.4 vs. 5.0, t=2.2, $p=0.03$), "MATURITY FEARS" (10.9 vs. 6.3, t=2,2, $p=0.03$), EAT-26 item "BULIMIA" (11.5 vs. 6.8, t=2.5, $p=0.01$), and higher GSI (1.9 vs. 1.3, t=2.2, $p=0.03$). They did not differ in the age at admission to the programme, age of illness onset, lifetime maximum or minimum weight and BMI, BMI at admission, and in the remaining EDI and EAT-26 items. There were also no significant differences in the sex, diagnosis, comorbidity, and type of treatment between the completers and non-completers.

In the whole group there was a significant effect of TIME in the repeated measures ANOVA with dependent variables EDI items "DRIVE FOR THINNESS", "BULIMIA", and "INTEROCEPTIVE AWARENESS", EAT-21 items "DIETING", "BULIMIA", and EAT-26 total score. There was a trend to statistical significance in the ANOVA designs with dependent variables EDI "BODY DISSATISFACTION" ($p=0.03$), "INEFFECTIVENESS" ($p=0.04$), "INTERPERSONAL DISTRUST" ($p=0.02$), "MATURITY FEARS" ($p=0.002$), EAT item "FOOD PREOCCUPATION AND ORAL CONTROL" ($p=0.01$), and GSI ($p=0.002$). All these significant results correspond to a decrease in the items between the baseline and end-point of the treatment.

Tab. 2. Sample characteristics according to the type of treatment.

	SSRI	No AD	Mirtazapine	Total
Females	40/41 (97.6%)	11/12 (91.7%)	29/31 (93.6%)	80
Males	1/41 (2.4%)	1/12 (8.3%)	2/31 (6.4%)	4
Bulimia	34/41 (83.0%)***	5/12 (41.7%)	2/31 (6.5%)	41
Anorexia	6/41 (14.6%)	4/12 (33.3%)	27/31 (87.0%)***	37
Atypical	1/41 (2.4%)	3/12 (25.0%)	2/31 (6.5%)	6
Completers	37/41 (90.2%)	10/12 (83.3%)	29/31(93.5%)	76
Non-completers	4/41 (9.8%)	2/12 (16.7%)	2/31 (6.5%)	8
No comorbidity	33/41 (80.5%)	8/12 (66.7%)	23/31 (74.2)	64
Comorbidity	8/41 (19.5%)	4/12 (33.3%)	8 /31 (19.5)	20
Total	41(48.8%)	12 (14.3%)	31 (36.9%)	84 (100%)

The table is a concatenation of 4 independent contingency tables with corresponding results of chi-quadrate tests: *** $p<0.001$ signif. more pts suffering from bulimia were treated with SSRI than pts suffering from anorexia and significantly more pts suffering from anorexia were treated with mirtazapine than pts suffering from anorexia

The effect of TREATMENT (AD, NO AD)

The distribution of the sample characteristics according to the type of treatment is presented in Table 2.

Seventy-two patients (85.7%) were treated with AD add-on. Four patients were treated with escitalopram, three citalopram, eleven sertraline, twenty paroxetine and three fluoxetine. Significantly more patients with bulimia than with anorexia were treated with SSRI and significantly more patients with anorexia than with bulimia were treated with mirtazapine.

Six (8.3% of all antidepressant-treated) patients treated with an antidepressant did not complete the treatment – in contrast to two (16.7% of patients without the add-on) without the antidepressant treatment; however, the results were not significant (chi-quadrate 0.8, $p=0.36$).

"TREATMENT" had no effect and there was no significant interaction between TREATMENT" and "TIME" parameters in the repeated measures ANOVA with any dependent variables.

Post-hoc analysis did not find significant differences in BMI. However, post-hoc analysis revealed significant differences ($p<0.001$) between baseline and end-point values of all but two EDI items ("INTERPERSONAL DISTRUST", "PERFECTIONISM"), all EAT-26 items and GSI and raw scores of dimensions "OBSESSIVE-COMPULSIVE ", "ANXIETY", "DEPRESSION" in the AD-treated group.

The effect of the treatment type

BMI – there was a significant effect of AD GROUP in the repeated measures ANOVA with dependent variable BMI and no interaction between "AD GROUP" and "TIME". Post-hoc analysis revealed significantly lower BMI at both time-points in patients treated with mirtazapine when compared with the patients treated with SSRI and without any antidepressant (Figure 1).

EDI – there was a significant effect of "AD GROUP" and significant interaction between "AD GROUP" and "TIME" on the "BULIMIA" score; post-hoc analysis revealed significantly higher baseline value in the group of patients treated with SSRI when compared with the mirtazapine treated group and a significant decrease between baseline and end-point again only in the group treated with SSRIs (see Figure 2).

For items "DRIVE FOR THINNESS", "BODY DISSATISFACTION" and "INTEROCEPTIVE AWARENESS" there was no effect of AD GROUP, no significant interaction; however, post-hoc analysis revealed significant differences ($p<0.001$) between baseline and end-point only in the group treated with SSRIs.

EAT – there was a significant effect of "AD GROUP" on the "FOOD PREOCCUPATION AND ORAL CONTROL" score; post-hoc analysis revealed a significantly higher value in the mirtazapine-treated group in contrast to patients treated with SSRI and patients without any antidepressant. Post-hoc analysis revealed significant differences ($p<0.001$) between baseline and end-point "BULIMIA" and total EAT-26 score only in the SSRI-treated patients corresponding to a decrease in these items (see Figure 3).

GSI and dimension raw scores – there was no effect of "AD GROUP" and no significant interaction between parameters on the SCL "OBSESSIVE-COMPULSIVE SYMPTOMS", "ANXIETY", "DEPRESSION" and global SCL score. Post-hoc analysis found significant differences ($p<0.001$) between baseline and end-point scores only for the SSRI-treated group with a corresponding decrease of GSI and raw scores for dimensions "OBSESSIVE-COMPULSIVE", " ANXIETY" and "DEPRESSION".

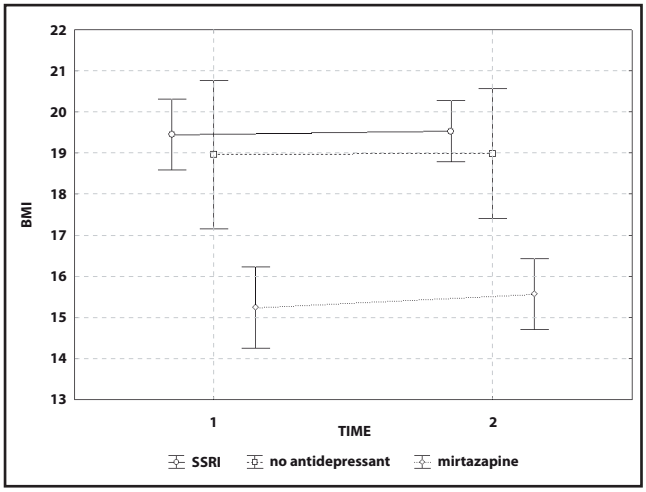

Fig. 1. Effect of AD group on BMI. BMI according to the antidepressant type: mirtazapine, SSRIs or no antidepressant, in two time points: 1 = baseline, 2 = end-point. Vertical bars represent 95% confidence intervals.

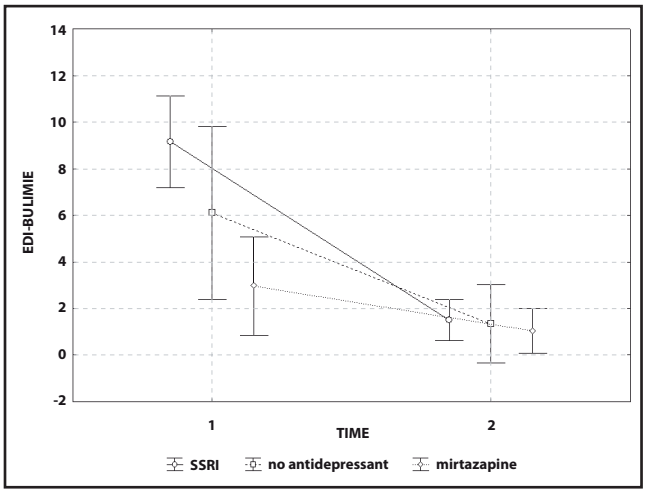

Fig. 2. Effect of AD group on EDI-BULIMIA score. BULIMIA score according to the antidepressant type: mirtazapine, SSRIs or no antidepressant, in two time points: 1 = baseline, 2 = end-point. Vertical bars represent 95% confidence intervals.

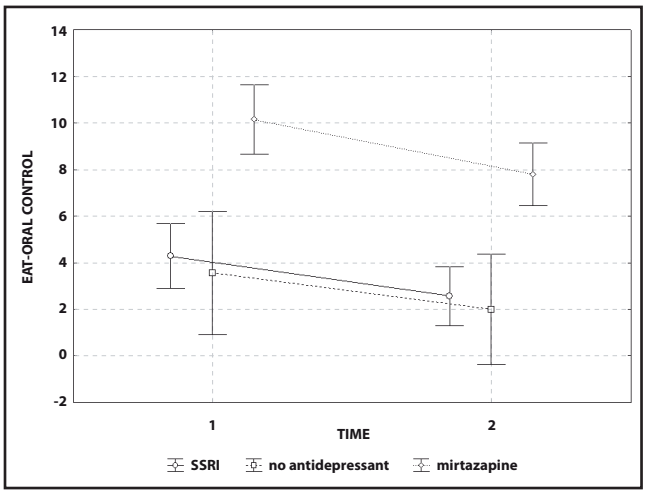

Fig. 3. Effect of AD group on EAT FOOD PREOCCUPATION AND ORAL CONTROL score. EAT ORAL CONTROL score according to the antidepressant type: mirtazapine, SSRIs or no antidepressant, in two time points: 1 = baseline, 2 = end-point. Vertical bars represent 95% confidence intervals.

DISCUSSION

The study has shown maintenance of anthropometric variables (BMI and weight) as well as an improvement of psychopathological variables in a group of patients with eating disorders on a special inpatient psychotherapeutic programme and targeted antidepressant treatment.

In the whole sample, in both bulimia and anorexia, the mean BMI remained stable. The difference between BMI in patients with AN and BN (e.g. significantly lower BMI in AN) has been expected and reflects the inherent differences in the clinical expression. Furthermore, an improvement in the attitudes and pathological behaviour concerning the eating habits (the decrease in EDI and EAT-26) and improvement in psychopathology, if present (decrease in GSI and dimension raw scores) were observed following the short-term intervention. Whether this improvement is maintained for a substantial period of time and thus if the change is clinically relevant will depend on a long-term outcome analysis, which we are planning to perform. Furthermore, a decrease in illness severity and subjective improvement of self-perception was reported. After 6 weeks of treatment, cooperativeness increased and thus the overall therapeutic process could advance in completers of the programme.

The ratio of non-completers to completers of the programme in our study (8/84) is lower than in published literature. This could be explained by the selection of the patients who entered the programme – not all patients on the waiting list turned up for hospitalization and our sample was biased by patients willing to cooperate. It is well know that individuals with AN are inherently difficult to treat, even in an inpatient setting. Moreover, it has been argued that inpatients with AN are more than twice as likely to drop out of an inpatient treatment programme than individuals in a general psychiatric ward (Kahn & Pike 2001; Zeeck *et al.* 2005).

Both the psychodynamic approach using imaging and targeted AD treatment enable comprehensive treatment on a more individualized basis. The Guided Affective Imagery is a successful psychotherapeutic technique in eating disorder (Wilke & Leuner 1990).

The effect of AD add-on was important in patients with detectable psychopathology. The choice of treatment was tailored to individual patients. We took into the consideration the diagnosis, subtype of disorder, individual psychopathology and previous experience and preference of the patients. The medication was given by nurses, so that compliance should be high. However, no objective measures of compliance have been used. There was a significant decrease of EDI, EAT-26 and selected SCL items irrespective of the presence of AD add-on. However, post-hoc analysis showed that this effect was mainly due to the AD-treated group: only patients treated with ADs showed a significant decrease in the items between baseline and end point

(with the exception of INTERPERSONAL DISTRUST AND PERFECTIONISM). This could be explained by an individual approach to the choice of treatment. Patients with low psychopathology usually had no additional pharmacotherapy. The unchanged INTERPERSONAL DISTRUST and PERFECTIONISM subscale scores may reflect a core personality feature in patients suffering from eating disorders, not influenced by treatment. Personality traits may constitute the intermediate phenotypes between genes and vulnerability to anorexia AN. It was found that polymorphisms in brain derived neurotrophic factor gene may influence the personality trait associated with higher risk of AN (Rybakowski *et al.* 2007). No significant differences in BMI were found. We tend to interpret these results as a beneficial effect of antidepressant treatment on the psychopathology of eating disorders, particularly on the secondary obsessive-compulsive, anxiety and depressive symptoms.

Concerning the effect of the AD treatment type (SSRI vs mirtazapine), patients treated with mirtazapine had significantly lower BMI at both time-points when compared with the patients treated with SSRI and without any antidepressants. This apparently reflects the fact that more anorexia nervosa patients were treated with mirtazapine, whereas bulimia patients were preferentially given SSRIs. Against expectations, the treatment with mirtazapine did not increase weight, but it succeeded in stabilizing weight, even in the patients with low BMI. Further, patients treated with SSRI had a significantly higher baseline EDI BULIMIA score value, and a subsequent significant decrease in this value, when compared with the mirtazapine treated group. This is understandable as only 2 of the 31 patients with bulimia were treated with mirtazapine and with mirtazapine the bulimia subscale score was the lowest. The same was true with 2 of 3 EAT-26 subscales BULIMIA AND FOOD PREOCCUPATION AND ORAL CONTROL. Interestingly, we also observed differences in the ability to influence the secondary psychopathology: obsessive-compulsive, anxiety and depressive symptoms were significantly lower only in the SSRIs treated patients.

Taken together, the observed differences in the mirtazapine and SSRIs effect on the psychopathology reflect the preferential use of these two groups of antidepressants in BN and AN respectively. Clinicians apparently tried to target the core clinical features of the eating disorders: SSRIs were given to bulimia patients with high scores of impulsivity, feature linked with the central serotonin activity. The frequent use of mirtazapine in the AN patients might reflect the clinical utilization of its adverse effect on weight – unfortunately, our data demonstrates that these expectations were not fulfilled.

ADs are not routinely used in the acute phase of treatment for severely malnourished pts. According to published literature, the medication should be considered in pts with AN for the prevention of relapse among weight restored patients or to treat associated features such as depression or obsessive-compulsive problems (in spite of or in the absence of weight gain) (APA 2000). The SSRIs are preferred because of their relative safety and tolerance in eating disorders. Several open trials have shown that fluoxetine improves outcome in people with AN (Kaye *et al.* 1991; Kim 2003); however, the results of two double-blind 1-year studies designed to determine whether a SSRI would improve outcome and reduce relapse after weight restoration were inconsistent (Kaye *et al.* 2001; Walsh *et al.* 2006). A MEDLINE literature search from 1966 through 2003 found that fluoxetine helped reduce symptoms of obsessive-compulsive disorder and depression in pts with AN (Kim 2003). Three studies support the efficacy of citalopram in treating AN, 2 open (Calandra *et al.*1999; Pallanti *et al.* 1997) and 1 double-blind study (Fassino *et al.* 2002). Recently case reports on successful treatment with mirtazapine were published in Czech psychiatric journals (Theiner & Zackova 2006). There is some limited experience with other dual ADs.

ADs are effective as one component of an initial treatment programme for most pts with BN. SSRIs are currently considered to be the safest antidepressants and may be especially helpful for pts with significant symptoms of depression, anxiety, obsessions, or certain impulse disorder symptoms or for those patients who have had a suboptimal response to previous attempts at appropriate psychosocial therapy (APA 2000). A review evaluated the use of ADs compared with placebo for the treatment of BN. Patients treated with tricyclic ADs dropped out due to any cause more frequently than patients treated with a placebo. The opposite was found for those treated with fluoxetine, suggesting it may be a more acceptable treatment. The use of a single AD was clinically effective for the treatment of BN when compared to a placebo, with an overall greater remission rate but a higher rate of dropouts. No differential effect regarding efficacy and tolerability among the various classes of ADs could be demonstrated (Bacaltchuk & Hay 2001). In a recent systematic review of randomized controlled trials, 47 studies of medication only, behavioural interventions only, and medication plus behavioural interventions were identified. Fluoxetine decreases core symptoms of binge eating and purging and associated psychological features in the short term; cognitive behavioural therapy reduces core behavioural and psychological features in the short and long term. The authors conclude that evidence for medication or behavioural treatment for BN is strong (Shapiro *et al.* 2007).

Our study does have limitations. The sample was biased by patients willing to cooperate, the study was performed under routine clinical conditions and our results cannot be generalized. Further, no markers of malnutrition (neuropeptides, hormones) were measured. This seems to be very important because an abnormal activity of neuropeptides may lead to disturbed control of appetite and hormonal dysregulation

in eating disorder (Baranowska *et al.* 2003). On the other hand, our comprehensive programme enables an individual approach, which seems to be important for programme accomplishment, subjective feeling and cooperation.

This prospective, open, naturalistic study confirmed a benefit from a comprehensive therapeutic approach to the treatment of patients with eating disorders. This approach is able to achieve weight stabilization, improvement in attitudes and pathological behaviour concerning the eating habits and improvement in psychopathology, if present. It means the choice of treatment tailored to the patient. Although the add-on of ADs exerts a certain beneficial effect on the symptomatology of eating disorders, the main role of antidepressant medication in the treatment of these conditions should be viewed as the influence on depressive, anxiety or obsessive-compulsive symptoms. However, the clinical relevance of this improvement needs to be confirmed in a long-term follow-up. Still, the pharmacological options for eating disorders, especially of AN are very limited and novel pharmacological interventions (Kmoch *et al.* 2009) are needed for each of these conditions.

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