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ORIGINAL ARTICLE

Risk factors for a low weight gain in the early stage of adolescent anorexia nervosa inpatient treatment: findings from a pilot study

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Abstract

Purpose Body weight restoration is a major treatment aim in juvenile inpatients with anorexia nervosa (AN) (i.e., 500–1000 g/week according to the German guidelines). Several studies suggest the early weight gain to be crucial for remission. The identification of patients at risk of a low early weight gain could enable an adequate adaptation of treatment. Thus, we aimed at detecting risk factors of a low weight gain during inpatient treatment.

Methods The presented work analyzes data from a pilot study in 30 female adolescent inpatients with AN (restricting subtype; age range at admission: 12.6–17.6 years). Premorbid characteristics, history of symptomatology, anthropometric data, and eating-disorder psychopathology were compared between those who gained at least an average of 500 g/week during the first 7 weeks of treatment (high weight gainers, HWG) and those who did not (low weight gainers, LWG).

Results At admission, LWG ($n = 15$) had a significantly higher BMI(-SDS) and scored significantly higher in the eating-disorder examination questionnaire (EDE-Q) than HWG ($n = 15$). A logistic regression analysis indicated both parameters to be independently associated with a low weight gain.

Conclusion Higher EDE-Q scores seem to be a major risk factor for a low weight gain at the beginning of treatment. Moreover, a higher BMI(-SDS) at admission does not necessarily indicate a less severe AN symptomatic, as it was associated with a lower weight gain in our sample during the first 7 weeks of treatment. Reassessment of our results in larger studies is required to draw firm conclusions for clinical practice.

Level of evidence Level V.

Keywords Anorexia nervosa · Body weight restoration · Body composition · EDE-Q

Introduction

Anorexia nervosa (AN) is a severe mental disorder, which is prone to chronification [1] and associated with an increased mortality risk [2]. AN mainly arises during adolescence [1] with prevalence rates ranging from 0.3 to 1.7% in adolescents [3, 4] and from 0.5 to 1% in adults [2]. A very low body weight and the fear of gaining weight are key features of AN [5] with potential consequences for the cardiovascular system, brain, endocrine system, bone mineral density, and linear growth [6]. Thus, one major treatment aim in children and adolescent AN patients is the restoration of a body weight to the normal range [7]. There is currently no superiority of any treatment setting for AN (outpatient, inpatient, reduced length of inpatient followed by outpatient, or partial hospital care); thus, among others, severity of illness

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or access to levels of care has to be taken into account when choosing an adequate treatment setting [8].

During inpatient treatment, the German guidelines (S3 guidelines) for diagnosis and treatment of eating disorders recommend an increase in body weight (BW) of 0.5–1 kg/week for adolescent patients with AN [9]. Other European countries also aim at weight gains of 0.5–1 kg/week to 0.5–1.5 kg/week [10]. According to the American Psychiatric Association (APA) guideline for patients with AN, hospitalized patients should gain 2–3 lb (0.9–1.4 kg) per week [11]. With respect to the initial energy intake, most guidelines seem rather conservative, and common recommendations for optimal refeeding practices are still lacking [7]. In published treatment approaches, patients mostly received low energy amounts at the beginning (~ 1200 kcal/day) which were slowly and gradually increased to prevent the refeeding syndrome [12–14]. More recently, safe and efficient weight restoration approaches were reported using higher caloric approaches and a close medical monitoring at the same time [15, 16]. For severe cases of AN, enteral nutrition is an essential life-saving treatment element [17]. Nevertheless, regarding its general use neither uniform protocols currently exist, nor does clarity on its benefits in the long-term or on recovery [17].

Several studies indicate that among both adolescent and adult patients with AN, early weight gain is a predictor for remission at the end of treatment [18, 19] and at follow-up [19–22]. Wales and colleagues found that adult AN inpatients were 18 times more likely to achieve a positive treatment outcome (i.e., a BMI of 17.5 kg/m^2 within an individual time frame) if they gained at least 500 g/week in the first 6 weeks of treatment [23]. In line with this, Hartmann and colleagues reported that a weight loss at the beginning of adult AN treatment was associated with a lower discharge BMI and a higher rate of unsuccessfully treated patients [24]. Hartmann and colleagues concluded that the identification of patients at risk of insufficient early weight gain could enable an adequate adaptation of the treatment process, and possibly result in higher treatment effectiveness [24].

A wide range of factors potentially influencing the early weight gain in different treatment approaches has been reported. Among adolescents with AN, lower parental education [18], the absence of psychiatric comorbidities [18], greater paternal or adolescent therapeutic alliance [25], lower paternal or maternal criticism [25] and less severe eating-disorder symptoms [25] were found to predict early weight gain. Among adolescents and young adults with (subthreshold) AN, Berona and colleagues found that rapid in contrast to slow responders had a lower BMI, used compensatory behaviors, but did not exhibit any mood or anxiety disorder at baseline [26]. Among adult women with AN, compensatory behaviors such as binging and vomiting [27], less severe eating-disorder symptoms [28], and a lower BMI [28] were associated with a faster/higher weight gain at the beginning of treatment.

Nevertheless, especially among adolescents with AN treated as inpatients, no solid predictors of early weight gain have been identified. Therefore, the aim of the present pilot study was to compare adolescent AN inpatients who achieved an average weight gain of at least 500 g/week—as recommended by the German and other European guidelines—during the first 7 weeks of treatment with those who did not. At this point, we focused on the premorbid characteristics, history of the symptomatology, anthropometric, and eating-disorder psychopathological characteristics.

Participants and methods

Participants and study schedule

The present analysis is a secondary analysis of data from a study cohort of 38 female adolescents with AN of the restricting subtype (age range on admission: 12.6–17.6 years) who were recruited between 2013 and 2015 in the Department of Child and Adolescent Psychiatry, Psychosomatics and Psychotherapy at the University Hospital Essen, Germany for metabolic and endocrine measurements. For the present analysis, we used anthropometric data and data on eating-disorder psychopathology collected at admission and at attainment of the defined target weight. In addition, information on routinely assessed parameters during the first 7 weeks (W0–W7) of inpatient treatment (i.e., individual body weight development, prescribed energy intake at admission, and nasogastric feeding) were extracted from patients' chart records (Fig. 1).

To compare patients who reached a target weight gain of at least 500 g/week and those with lower weight gain, we decided to focus on the first 7 weeks of inpatient treatment. In general, there are inter-individual differences in the duration of inpatient treatment with a mean duration of 3.4 months in Germany [29]. The examined period thus approximately represents the first half of the average duration for inpatient treatment. Full information on weight development during the first 7 weeks of treatment was available for 30 patients, which were included in the present analysis.

Psychiatric diagnoses were ascertained via clinical examination and a structured interview (DIA-X/M-CIDI; [30]) according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR, [31]). The target weight was individually defined according to the applying German guidelines [9] within the range of the 10th and 25th BMI percentile and/or according to the premorbid course of the BMI percentile and/or the lack of somatic complications. All patients were treated as

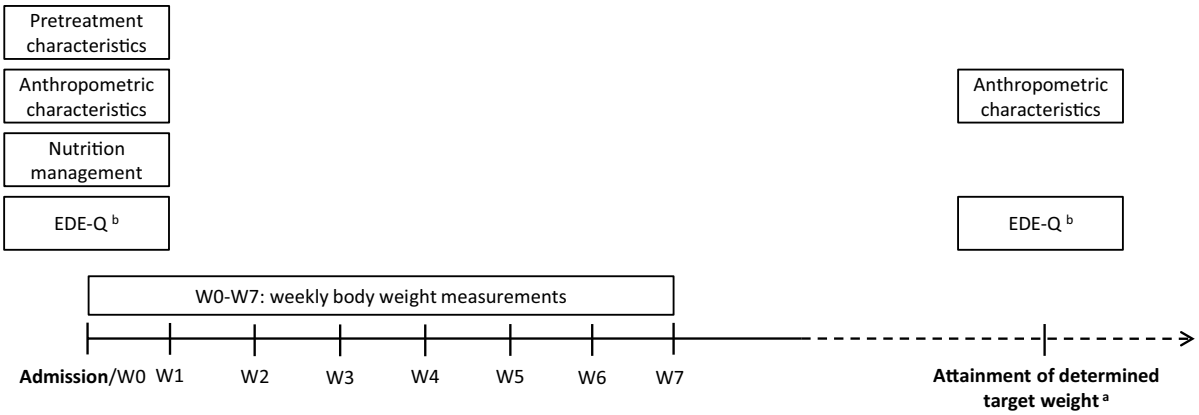

Fig. 1 Study schedule and variable assessments. Anthropometric characteristics (BMI, BMI-SDS, and body composition) and eating-disorder psychopathological data (via EDE-Q) were determined at admission and at attainment of the defined target weight. Data on body weight development during the first 7 weeks (W0–W7) and information on nutrition management (i.e., prescribed energy intake at admission and requirement of nasogastric feeding during treatment) were extracted from patients' records. [a]An individual target weight was defined at the beginning of treatment for each patient. The target weight was reached in 63–618 days (median: 144 days). After W7, most of the patients were treated as inpatients until having reached the defined target weight, some as daycare or outpatients. [b]*EDE-Q* eating-disorder examination questionnaire

inpatients at least until W7, and most of them remained inpatients and/or day patients until having reached the defined target weight. If patients did not attain their target weight within the in-/day patient treatment, they were asked to attend a follow-up assessment as soon as their target weight was reached in the outpatient setting.

Therapeutical and nutritional approach

The multimodal treatment program of the inpatient unit is based on weight restoration, medical observation, cognitive–behavioral therapy, family therapy, and nutritional counselling. For each patient, a positive fortifier plan based on BW gain in 500 g steps was established on an individual basis. In line with the applying German guidelines [9], prescribed diets contained 30–40 kcal/kg BW/day at the beginning of treatment. Nursing stuff supervised food intakes at breakfast, lunch, and dinner. Nasogastric feeding was recommended during realimentation if the severity of psychopathology and eating behavior hampered the patient consistently to gain weight sufficiently.

Anthropometric measurements

All anthropometric measurements were performed between 7 a.m. and 9 a.m. after overnight fasting. At admission and at attainment of the target weight, body weight and height were measured using the same calibrated electronic scales and stadiometers. Patients were weighed in underwear. BMI standard deviation scores (BMI-SDS) were calculated based on German reference data for children and adolescents [32] using the LMS method [33]. Information on fat-free mass (FFM in kg and %) and fat mass (FM in kg and %) was derived from air displacement plethysmography using the thoracic gas volume method in the BodPod (Life Measurement, Inc., Concord, CA, USA) [34] and software V4.2+ as supplied by the manufacturer (Life Measurement Inc., Concord, CA, USA). BodPod measurements were conducted in underwear with metal objects (e.g., watches) removed. Patients wore a cap and had to empty their bladder before measurement. Physical activity was not allowed 2 hs prior to measurements [35].

Eating-disorder psychopathology and history of symptomatology

At admission and at attainment of the target weight, individual eating-disorder psychopathology was examined with the German version of the Eating-Disorder Examination Questionnaire (EDE-Q) [36]. The EDE-Q [37] is a 36-item self-report measure adapted from the structured "Eating-Disorder Examination" interview (EDE) [38]. Similar to the EDE, the EDE-Q spans a 28-day time frame, yields four subscales, and is scored on a seven-point Likert scale. It measures severity of eating-disorder psychopathology with four subscales (eating concern; weight concern; shape concern; and dietary restraint), one global score, and ED behaviors. Items comprising the global scale and subscales are rated on a 0–6 scale, with higher scores reflecting greater severity. The German version of the EDE-Q has been validated in patient samples of AN ($n = 105$), bulimia nervosa ($n = 55$), atypical eating disorders ($n = 54$) as well as non-eating-disordered participants ($n = 409$); internal consistencies between subscales and total score were high (subscales:

0.85 ≤ Cronbach's $\alpha \leq 0.93$; total score: Cronbach's $\alpha = 0.97$) [36]. In addition, duration of illness (in months) was assessed at admission using a self-designed questionnaire. Duration of illness was defined as lapse of time between recalled initiation of weight loss or insufficient age-appropriate weight gain or occurrence of secondary amenorrhea (whatever appeared first) and admission. AN pretreatments (inpatient/outpatient) were also recalled.

Ethics statement

The study was approved by the ethics committee of the University Hospital Essen and followed the Declaration of Helsinki. All participants and their parents provided written informed consent.

Statistics

Mean weight gain per week was calculated based on the eight weekly BW measurements (W0–W7; W0 = BW during the first week after admission). Patients were divided into two groups: (1) mean body weight gain of at least 500 g/week (high weight gainers, HWG) or (2) body weight gain below 500 g/week (low weight gainers, LWG). The threshold of 500 g was chosen, as it represents the minimum expected weight gain per week for AN inpatients according to the applying German guidelines [9].

All statistical tests were performed using SAS® procedures (version 9.2, Statistical Analysis Systems, Cary, NC, USA). In a first step, differences between HWG and LWG with regard to premorbid characteristics, history of symptomatology and characteristics at admission were explored. As graphically tests revealed that nearly all parameters of interest were non-normally distributed, it was decided to uniformly calculate medians as well as first and third quartiles for continuous parameters and to analyze differences between HWG and LWG using Wilcoxon rank sum test. Considering the small sample size, exact p values were computed. Fisher's exact test was used for categorical parameters. In a second step, variables identified in unifactorial testing to significantly differ between the two groups at admission, i.e., EDE-Q total score and BMI-SDS, were included as exposures in a logistic regression model (using the enter method). Group affiliation, i.e., HWG or LWG, was defined as outcome variable. BMI-SDS instead of BMI was chosen as exposure to calculate age-independent effect estimates. Results were presented as odds ratios (OR) and 95% confidence intervals (95% CI). For all statistical tests, p values < 0.05 were considered significant. A correction for multiple testing was not performed due to the exploratory character of this pilot study and the small sample size.

Results

Half of the 30 participants included in the present analyses gained an average of at least 500 g BW per week until W7 (HWG, Table 1). The weight gain in the HWG was consistently higher in both the first (W0–W3) and the second half (W3–W7) of the evaluated period compared to the LWG group (Table 1).

Characteristics at admission

At admission, major comorbid diagnoses were depressive disorder (20%) followed by generalized anxiety disorder (17%) and somatoform disorders (17%). General prevalence of comorbid diagnoses did not differ between HWG and LWG. BMI and BMI-SDS were significantly lower in the HWG compared to the LWG and the HWG scored significantly lower in the EDE-Q total score as well as in each sub-score (Table 2). Total duration of illness before admission did not differ between the two groups. The setting of pretreatment tended to differ with a higher percentage of previous outpatient treatment in the HWG and a higher percentage of the previous inpatient treatment in the LWG (Table 2). Prescribed energy intake differed neither in total amount nor in relation to individual body weight at admission. During treatment, two HWG and eight LWG required nasogastric feeding (Table 2).

Positive ORs deriving from the logistic regression analysis confirm that both a high BMI-SDS and high EDE-Q total scores at admission are independently associated with a low weight gain (Table 3). P values of both variables were

Table 1 Weight gain during the first 7 weeks of treatment

Variable	High weight gainers (HWG)		Low weight gainers (LWG)		p^b
	n^a	Median (P25; P75)	n^a	Median (P25; P75)	
Weight gain W0–W7 (kg/week)	15	0.57 (0.53; 0.79)	15	0.29 (0.20; 0.41)	$\mathbf{1.3 \times 10^{-8}}$
Weight gain W0–W3 (kg/week)	14	0.58 (0.37; 0.77)	15	0.17 (− 0.23; 0.40)	$\mathbf{1.6 \times 10^{-4}}$
Weight gain W3–W7 (kg/week)	14	0.71 (0.55; 0.78)	15	0.38 (0.28; 0.58)	$\mathbf{7.5 \times 10^{-4}}$

[a]Number of subjects with available data

[b]Differences were tested using Wilcoxon rank sum test (option exact p values), bold values represent significant differences between the two groups; P25, 25th percentile; P75, 75th percentile

Table 2 Premorbid characteristics, history of symptomatology and characteristics at admission, 7 weeks after admission and at attainment of the defined target weight

Variable	HWG		LWG		r^b	p^c
	n^a	Median (P25; P75)	n^a	Median (P25; P75)		
Premorbid characteristics						
Age at onset of AN (years)	13	14.4 (13.2; 15.4)	14	14.2 (13.1;15.2)	0.07	0.720
Body weight (kg)	15	53.0 (44.5; 58.0)	15	52.0 (46.0; 58.0)	−0.07	0.705
BMI (kg/m^2)	15	20.0 (17.2; 21.2)	15	19.7 (18.1; 20.7)	0.02	0.903
BMI-SDS[d]	13	−0.45 (−1.37; 0.01)	14	−0.29 (−0.76; 0.00)	−0.06	0.756
BMI percentile[d]	13	32.5 (8.5; 50.4)	14	38.7 (22.3; 50.2)	−0.06	0.756
History of symptomatology						
Duration of illness (months)	15	8 (7;12)	12	8 (5.5; 16.5)	−0.08	0.673
No pretreatment [*n* (%)]	15	3 (20)	13	2 (15)	n.a.	0.076
Outpatient pretreatment [*n* (%)]	15	9 (60)	13	3 (23)		
Inpatient pretreatment [*n* (%)]	15	3 (20)	13	8 (62)		
Characteristics at admission/W0						
Age and anthropometric parameters						
Age (years)	15	15.5 (14.1; 16.4)	15	15.2 (13.9; 16.8)	−0.04	0.838
Body weight (kg)	15	40.1 (36.3; 44.9)	15	40.1 (37.2; 46.5)	0.11	0.533
Height (cm)	15	164 (161; 170)	15	161 (156; 165)	−0.17	0.349
BMI (kg/m^2)	15	14.7 (13.7; 15.5)	15	15.6 (14.9; 16.7)	0.47	**0.009**
BMI-SDS[d]	15	−3.5 (−4.0; −2.6)	15	−2.1 (−3.3; −2.0)	0.48	**0.008**
BMI percentile[d]	15	0.02 (0.00; 0.52)	15	1.83 (0.05; 2.24)	0.48	**0.008**
Individually defined target BMI (kg/m^2)	15	18.7 (17.4; 19.5)	15	18.7 (18.5; 19.3)	0.07	0.705
FFM (kg)	15	36.5 (32.8; 39.6)	12	37.7 (34.8; 41.3)	0.16	0.399
FM (kg)	15	1.8 (1.3; 6.4)	12	3.7 (2.0; 6.2)	0.09	0.624
FM (%)	15	5.6 (3.2; 14.8)	12	8.2 (4.9; 15.0)	0.08	0.674
Eating-disorder psychopathology						
Total EDE-Q	15	2.7 (1.7; 4.0)	13	4.5 (4.0; 5.3)	0.49	**0.008**
Eating concern	15	2.8 (0.8; 3.6)	13	4.2 (3.2; 4.6)	0.49	**0.008**
Body weight concern	15	2.8 (1.0; 4.0)	13	5.2 (3.6; 5.8)	0.48	**0.009**
Shape concern	15	3.8 (2.1; 4.9)	13	5.3 (4.6; 5.9)	0.41	**0.026**
Restraint	15	3.2 (1.4; 4.2)	13	5.4 (3.4; 5.6)	0.39	**0.037**
Nutrition management						
Prescribed energy intake (kcal/d)	12	1350 (1100; 1700)	15	1350 (1200; 1500)	−0.01	0.932
Prescribed energy intake (kcal/kg BW/d)	12	34.8 (23.5; 45.8)	15	32.7 (27.5; 39.8)	0.005	0.981
Nasogastric feeding [*n* (%)][e]	15	2 (13)	15	8 (53)	n.a.	0.050
Characteristics 7 weeks after admission (W7)						
Body weight (kg)	15	44.7 (40.1; 48.6)	15	43.2 (38.7; 48.1)	−0.03	0.846
BMI (kg/m^2)	15	16.2 (15.4; 17.0)	15	16.7 (15.9; 17.1)	0.14	0.436
BMI-SDS[d]	15	−2.2(−2.5; −1.7)	15	−2.0 (−2.5; −1.3)	0.14	0.436
BMI percentile[d]	15	1.58 (0.63; 4.02)	15	2.05 (0.63; 9.84)	0.14	0.436
Characteristics at attainment of the defined target weight						
Age and anthropometric parameters						
Age (years)	10	16.0 (14.3; 17.0)	9	15.7 (14.1; 17.2)	−0.10	0.661
Body weight (kg)	10	51.2 (46.5; 53.9)	9	47.5 (44.9; 52.3)	−0.18	0.447
BMI [kg/m^2]	10	18.4 (17.7; 19.0)	9	18.6 (17.6; 19.2)	0.03	0.905
BMI-SDS[d]	10	−1.06 (−1.55;−0.96)	9	−0.99 (−1.23;−0.69)	0.20	0.400
BMI percentile[d]	10	14.4 (6.1; 16.8)	9	16.1 (10.9; 24.4)	0.20	0.400
FFM (kg)	10	38.8 (36.9; 43.2)	9	39.3 (37.7; 45.6)	0.16	0.497
FM (kg)	10	10.8 (9.5; 14.7)	9	8.1 (5.9; 9.8)	−0.46	**0.044**
FM (%)	10	21.7 (19.2; 26.8)	9	16.0 (13.0; 20.7)	−0.51	**0.023**

Table 2 (continued)

Variable	HWG		LWG		r^b	p^c
	n^a	Median (P25; P75)	n^a	Median (P25; P75)		
Eating-disorder psychopathology						
Total EDE-Q	10	1.9 (1.0; 4.3)	9	3.4 (1.3; 4.3)	0.18	0.434
Eating concern	10	0.8 (0.4; 4.0)	9	3.2 (1.0; 3.8)	0.12	0.587
Body weight concern	10	2.3 (1.2; 4.4)	9	4.4 (1.4; 4.8)	0.21	0.366
Shape concern	10	3.5 (1.5; 5.0)	9	5.3 (2.0; 5.5)	0.18	0.433
Restraint	10	1.0 (0.4; 2.6)	9	1.6 (0.8; 2.8)	0.30	0.187
# of days to attain defined target weight	10	148 (105; 422)	9	144 (97; 165)	−0.14	0.549
Weight gained (kg)	10	9.3 (7.8; 10.8)	9	6.6 (5.8; 9.0)	−0.40	0.079

Data from female AN inpatients with a high weight gain (on average ≥ 500 g/week; HWG) were compared to those with a low weight gain (LWG)

AN Anorexia nervosa; *BMI(-SDS)* body mass index (standard deviation score), *BW* body weight, *EDE-Q* eating-disorder examination questionnaire, *HWG* high weight gainers, *LWG* low weight gainers; *P25* 25th percentile, *P75* 75th percentile

[a]Number of subjects with available data

[b]Calculated as $r = \frac{Z}{\sqrt{N}}$ with Z values deriving from Wilcoxon rank sum test

[c]Differences were tested using Wilcoxon rank sum test (option exact p values) for continuous parameters and Fisher's exact test for categorical parameters, bold values represent significant differences between the two groups

[d]SDS values and percentiles calculated using German Health Interview and Examination Survey for Children and Adolescents (KiGGS) data [32] as reference data

[e]Nasogastric feeding was recommended during realimentation if the severity of eating-disorder psychopathology and eating behavior hampered the patient consistently to gain at least 500 g/week of BW

Table 3 Results from logistic regression analysis on eating-disorder psychopathology and BMI-SDS on admission as potential risk factors of a low weight gain (LWG) in the first 7 weeks of treatment of female AN inpatients ($n = 28$)

Determinant	OR (95% CI)	*P*
EDE-Q total score at admission	2.532 (1.061;6.041)	**0.0362**
BMI-SDS at admission	3.045 (0.922;10.059)	0.0679

BMI-SDS and EDE-Q total score at admission were concomitantly included as exposition variables in one model to adjust for potential bidirectional interrelationships; Odds ratios > 1 indicate an increased risk of low weight gain (i.e., < 500 g/week) in the first 7 weeks

OR odds ratio, *95% CI* 95% confidence intervals, bold values represent significant differences between the two groups

attenuated compared with the unifactorial analyses and no longer significant for BMI-SDS.

Characteristics at W7 and at attainment of the defined target weights

At W7, BMI and BMI-SDS no longer differed between groups (Table 2). At attainment of the defined target weight, follow-up data were available for only ten HWG and nine LWG. EDE-Q total scores as well as scores of each subscale improved during treatment in both groups. Although absolute values still seem to be higher in the LWG, there was no statistically significant difference between both groups at attainment of the defined target weight. Moreover, neither BMI nor BMI-SDS differed, but HWG had a significantly higher FM compared to the LWG (Table 2).

Discussion

Early weight gain in the treatment of patients with AN is discussed as a predictor of a successful treatment outcome [18–24]. In the present study, only 50% ($n = 15$) gained at least 500 g per week during the first 7 weeks of inpatient treatment (HWG). The main finding was that at admission, the LWG had a higher BMI and BMI-SDS, but also higher EDE-Q scores indicating a more severe eating-disorder psychopathology compared to the HWG. A multifactorial model that included both BMI-SDS and EDE-Q as exposure indicated that both might be independent risk factors of an early insufficient weight gain.

The LWG were characterized by a more severe eating-disorder psychopathology measured by the EDE-Q (total score: 4.5 vs. 2.7). Thus, despite a higher (i.e., less alarming) BMI at admission, the LWG were more concerned with their eating habits, body weight, and shape and more restrictive in their eating attitudes compared with the HWG. This is in line with another study among adult patients with AN: a higher BMI and a higher EDE-Q global score at baseline

were associated with a lower weight gain in the first six sessions of an outpatient treatment [28]. Consistently, adolescent inpatients with a higher BMI and more severe eating-disorder-specific symptoms were less motivated to change at admission and had a lower weekly BW gain during treatment [39]. More specifically, the EDE-Q sub-score "restraint" was found to be negatively correlated with the caloric intake of a test meal among adult patients [40]. The patients of that study scored with 4.29 on the restraint scale and thus showed even less restraint than the LWG of our study (5.4 vs. 3.2 in the HWG). When we additionally consider that dietary restraint seems to be strongly related to (deep-rooted) body image concerns [41], it is very reasonable to assume that the LWG in our study might have had more problems to achieve a dietary intake as prescribed at the beginning of treatment. This most likely explains the higher rate of nasogastric feeding during treatment in this group (8 vs. 2), as this approach was recommended in case a patient consistently struggled to gain weight sufficiently.

The fact that more than half of the LWG (62% vs. 20% among the HWG) had received at least one previous inpatient treatment might also explain the higher BMI(-SDS) in this group. One might assume that parents and/or the outpatient treatment system are more alert to repeated weight losses in these patients and that re-admission is arranged before weight loss reaches extreme extents. Therefore, in spite of the higher BMI(-SDS), these patients with a history of more intensive treatment were probably the more severe cases, which is confirmed by the higher EDE-Q scores and the result that more of them required nasogastric feeding.

One further explanation for the different degrees of weight gain during the first 7 weeks of treatment could be different states of starvation and differing degrees of a reduction in metabolic rate as metabolism is downregulated during chronic energy deficiency [42]. Resting energy expenditure was not assessed in the current study. However, a lower BMI at admission—as found among the HWG—might indicate a more severe state of starvation accompanied by a lower metabolic rate. Consequently, HWG might have benefitted more from the same amount of energy intake than the LWG, due to lower requirements. Indeed, a lower BMI was associated with a higher weight gain per excess calorie in young adult patients with AN [43].

Another possible reason for the higher weight gain in the group with the initially lower BMI may be a different perception of severity in the attending team. Restoration of a low body weight to the normal range represents a high treatment priority in juvenile patients with AN because of the severe implications of starvation for health [7]. It might be hypothesized that the lower the BMI(-SDS) at admission, the more efforts are made by medical and/or nursing staff to support realimentation. Therefore, eating and exercise behavior of patients with a lower BMI(-SDS) as found in the HWG might have been monitored more intensively by the clinical team.

At attainment of the defined target weight, neither BMI(-SDS) nor EDE-Q sores between both groups differed anymore significantly between both groups, although the latter still appear to be descriptively higher among the LWG (e.g., total EDE-Q score: 3.4 vs. 1.9 in the HWG). Although this might indicate a still more severe eating-disorder psychopathology, our study did not have sufficient statistical power to examine significant differences at this timepoint.

Strengths and limitations

To the best of our knowledge, our pilot analysis is the first one evaluating anthropometric, eating-disorder psychopathological and pretreatment characteristics at admission between adolescent inpatients with a weight gain of at least 500 g/week (as recommended by several European guidelines) and those with a lower weight gain during the first 7 weeks of treatment.

However, the evidence of the differing characteristics obtained is limited by the small sample size. This clearly results in a loss of statistical power, so that further differences between HWG and LWG could be identified in similar examinations with larger sample size. In addition, findings from this small study group might not be generally transferable to other patient groups or settings. Future studies on larger sample sizes are needed to further examine the hypotheses set by the current work. These should include metabolic assessments (i.e., resting energy expenditure, physical activity) as well as measurements of body composition that allow for differentiation of FFM (e.g., DXA). Moreover, for the present analysis, no follow-up data were available after the defined target weight was attained. As many patients with AN are vulnerable to relapse, follow-ups should certainly be included in future works to evaluate whether the treatment was successful in the long term. In this context, it would be interesting to include more complex body weight restoration patterns, for instance, the stability/non-stability (i.e., linearity/non-linearity) of the BW curve in the beginning of treatment, as recently suggested by Avnon and colleagues [44].

Conclusion

Comparing patients with a weight gain of at least 500 g/week on average and those with lower gain during the first 7 weeks of treatment our analysis revealed that higher scores in the EDE-Q seem to be a major risk factor for an insufficient weight gain at the beginning of the treatment. Moreover, a higher BMI(-SDS) on admission does not necessarily

indicate a less severe AN symptomatic and could—at least in our sample—even be a determinant of a slower and less efficient weight restoration. Moreover, due to the small sample size, our data need to be reassessed in larger study cohorts, so that firm conclusions for clinical practice can be drawn. In addition, these studies should also consider further important potential determinants such as resting energy expenditure.

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Compliance with ethical standards

Conflict of interest On behalf of all authors, the corresponding author states that there is no conflict of interest.

Ethical standards All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent In our study, all participants (and their parents) provided informed consent prior to their participation.

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