ABSTRACT

Background: Although the effect of immediate weight restoration on body composition and body fat distribution has previously been studied in anorexia nervosa (AN), its influence in women with AN on eating disorder psychopathology and psychological distress has not previously been investigated to our knowledge.

Objectives: We assessed body composition and fat mass distribution before and after body weight restoration and investigated any relation between changes body fat patterns of patients with AN treated in a specialist inpatient unit and their eating disorder and psychological distress features.

Design: Body composition was measured by using dual-energy X-ray absorptiometry in 50 female, adult patients with AN before and after complete weight restoration [body mass index (BMI; in kg/m²) ≥18.5] and 100 healthy control subjects matched by age and post-treatment BMI of study group participants. Eating disorder psychopathology and psychological distress were assessed in the AN group before and after weight restoration by using the Eating Disorder Examination interview and the Global Severity Index of the Brief Symptom Inventory (BSI-GSI), respectively.

Results: After the achievement of complete weight restoration, patients with AN had higher trunk (P < 0.001), android (P < 0.001), and gynoid (P < 0.001) fat masses and lower arm (P < 0.001) and leg (P = 0.001) fat masses with respect to control subjects. No relation was shown between body-composition variables and eating disorder psychopathology in the AN group, and the only significant predictor of change in BSI-GSI was the baseline BSI-GSI score.

Conclusion: The normalization of body weight in patients with AN is associated with a preferential distribution of body fat in central regions, which does not, however, seem to influence either eating disorder psychopathology or psychological distress scores. This trial was registered at www.controlled-trials.com as ISRCTN922626057.


INTRODUCTION

Anorexia nervosa (AN) is characterized by a restriction of energy intake relative to that required, which causes sufferers to become significantly underweight (1). Both the symptoms associated with low body weight and dietary restriction (starvation symptoms) and the psychopathology behind the eating disorder (2) can be addressed through nutritional rehabilitation (3), weight restoration, and cognitive behavioral therapy (4). These strategies are also necessary to restore an adequate quantity of body mass, both lean and fat, to enable physiologic processes (ie, a menstrual cycle and resting energy expenditure) to resume (5).

As to be expected from their undereating and underweight conditions, a marked reduction in both fat and lean masses in individuals with AN has been well documented (6, 7). Several studies have examined changes in body composition and body fat distribution in adults with AN. Of these studies, the majority of trials showed a preferential trunk fat accumulation associated with an acute weight regain immediately after treatment (8–11), although this was not a universal finding (12). Longer term, one study showed no differences in visceral adipose tissue distribution between former patients with AN who had maintained normal weight for 1 y and BMI-matched control women with no history of AN (13). However, a lower percentage of body fat restoration after weight restoration does seem to be a risk factor for relapse in patients with AN (14, 15).

Nevertheless, the research conducted to date on this issue has suffered from various methodologic limitations, such as small sample groups (8, 9), incomplete weight restoration (8, 9), and small (8, 10, 11, 13) and unsuitable control groups (normal-weight but not lean participants) (12). Thus far, research has also failed to address the relation between fat distribution, eating disorder psychopathology, and the psychological distress associated with weight restoration in AN (16). Indeed, although some authors have hypothesized that patients with greater trunk fat deposition after weight restoration could experience considerable stress brought on by changes in their body shapes, which make them more prone to a relapse (10), data to support this theory are lacking. A reported association between depressive mood and visceral adipose tissue in overweight women (17), possibly involving the leptin-mediated dysregulation of the

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4Abbreviations used: AN, anorexia nervosa; BMC, bone mineral content; BSI, Brief Symptom Inventory; BSI-GSI, Global Severity Index of the Brief Symptom Inventory; CBT-E, enhanced form of cognitive behavioral therapy; DXA, dual-energy X-ray absorptiometry; EDE, Eating Disorder Examination interview; GSI, Global Severity Index.

Received October 25, 2013. Accepted for publication January 21, 2014. doi: 10.3945/ajcn.113.078816.
In this context, as in other settings, dual-energy X-ray absorptiometry (DXA) is becoming an increasingly useful method of measuring body composition (19). However, previous DXA studies conducted on this issue have only examined certain patterns of body fat (i.e., legs, arms and, trunk) with respect to total fat mass (i.e., legs:total mass; arms:total mass and trunk: total mass), and to our knowledge, no previous study has examined android or gynoid patterns in individuals with AN. Such an investigation would enable a more-accurate assessment of abdominal fat because android fat seems significantly associated with the clustering of metabolic syndrome components (20). It would also be of interest to compare such data with those of control subjects matched by age and patient posttreatment BMI.

Therefore, we set out to 1) assess the quantity and distribution of lean and fat masses before and after complete weight restoration in patients with AN and 2) compare the body composition and fat distribution of weight-restored patients with those of healthy controls matched by age and patient posttreatment BMI. We also aimed to assess any relation between body-composition patterns and eating disorder psychopathology or general distress in patients with AN.

**SUBJECTS AND METHODS**

**Participants**

Fifty white, female patients with AN and 100 normal-weight, white, female subjects were enrolled in the study. All 50 patients were voluntarily and consecutively admitted to the eating disorder inpatient unit of Villa Garda Hospital during the years 2011–2013. Inclusion criteria were as follows: 1) aged 18–50 y, 2) BMI (in kg/m²) ≥17.5, 3) Eating Disorder Examination interview (EDE; 12th ed). The inpatient treatment has been described in detail elsewhere (23). In brief, the treatment was an adapted version of the enhanced form of cognitive behavioral therapy (CBT-E) for eating disorders (4) that lasts 20 wk and comprises 13 wk of inpatient therapy followed by 7 wk of partial hospitalization. The program retained all main strategies and procedures of the CBT-E, which are delivered in both individual and group sessions. However, the program differed from the outpatient CBT-E in that patients received dietitian-assisted eating in the early weeks of treatment until their BMI reached ≥18.5. Over the first 3 wk of treatment, the dietary intake of patients was increased incrementally from 1500 to 2500 kcal. In subsequent weeks of the program, the dietary energy content was adjusted to maintain a weight gain rate from 1 to 1.5 kg/wk until the BMI of patients reached ≥19. Once this target was achieved, the calorie content of meals was continually adjusted so that body weight of patients remained within a 2-kg range of this figure. The diet included all main food groups and conformed to Italian National Guidelines for Healthy Eating (24).

**Inpatient treatment protocol**

In addition, patients with BMI ≥15 and stable medical conditions participated in physiotherapist-led 60-min physical exercise sessions 2 times/wk as part of their treatment. These sessions included calisthenic exercises to help restore muscle strength, flexibility, and posture as well as aerobic exercises to improve cardiovascular fitness. No psychotropic medication was prescribed as part of the treatment, and any psychotropic drugs (i.e., selective serotonin reuptake inhibitors) that were being taken at admission (by 8 patients) were gradually phased out during the first 2 wk of hospitalization.

**Methods**

All data were collected in the first week of inpatient admission and during the last week of the program.

### Table 1

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Pretreatment AN (n = 50)</th>
<th>Control group (n = 100)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>26.5 ± 7.6^2</td>
<td>26.5 ± 7.6</td>
</tr>
<tr>
<td>Highest educational qualification</td>
<td></td>
<td></td>
</tr>
<tr>
<td>[n (%)]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Elementary school</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Middle school certificate</td>
<td>13 (26.0)</td>
<td>0</td>
</tr>
<tr>
<td>High school diploma</td>
<td>26 (52.0)</td>
<td>50 (50.0)</td>
</tr>
<tr>
<td>Bachelor’s degree</td>
<td>9 (18.0)</td>
<td>50 (50.0)</td>
</tr>
<tr>
<td>Missing data</td>
<td>2 (4.0)</td>
<td>0</td>
</tr>
<tr>
<td>Presence of amenorrhea [n (%)]</td>
<td>42 (84.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Duration of illness (y)^3</td>
<td>8.7 ± 6.7</td>
<td>—</td>
</tr>
<tr>
<td>Amount of exercise (min)^3,4</td>
<td>1533.9 ± 2732.3</td>
<td>—</td>
</tr>
</tbody>
</table>

^2 AN, anorexia nervosa.  
^3 Mean ± SD (all such values).  
^4 Data not available for the control group.

^5 In the past 28 d (measured by using the Eating Disorder Examination interview, 12th ed).
Body weight and height

Body weight and height were measured by a medical doctor by using medical weighing scales and a stadiometer, respectively. Participants were weighed before breakfast wearing only underwear and no shoes. The BMI of patients was determined according to the standard formula of body weight measured in kilograms divided by height in meters squared.

Body composition and body patterns

DXA scans [Prodigy Primo Lunar, A223040501; General Electric Company; USA-EnCORE TM 2009 (v13.31) software; General Electric Company] were used to measure total and regional fat, lean, and bone masses. Scans were performed in the morning, and no special preparations were made except for instructing participants to wear only underwear and no metal accessories.

Fat masses were measured in the arm, trunk, leg, and android and gynoid regions. The upper limit of the arm region was a hypothetical line from the crease of the axilla through the glenohumeral joint. The trunk region comprised the neck, chest, abdomen, and pelvis and extended from the inferior edge of the chin to a line that intersected the center of the femoral necks (below the pelvic brim). The leg region was taken to include all anatomic structures below this line. Within the trunk region, the android region was taken as the area between the ribs and the pelvis with an upper demarcation that was 20% of the distance between the iliac crest and neck and a lower perimeter that coincided with the top of the pelvis. The gynoid region, which comprised the hips and upper thighs, overlapped parts of both the legs and trunk; the upper demarcation of this region fell below the top of the iliac crest at a distance 1.5 times the android height. The total height of the gynoid region was twice that of the android region.

More details concerning regional body-composition analysis has been described elsewhere (25, 26). In this study, we assessed the following variables:

1) Total body composition, which included
   a) Total fat mass (kg)
   b) Fat mass as a percentage of the total mass (%), which was calculated as
   \[
   \frac{\text{Total fat mass}}{\text{Total fat mass} + \text{Total lean mass} + \text{Total BMC}} \times 100
   \]  
   (1)
   c) Fat mass index (kg/m²)
   d) Total lean mass (kg)
   e) Lean mass as a percentage of total mass (%), which was calculated as
   \[
   \frac{\text{Total lean mass}}{\text{Total lean mass} + \text{Total BMC}} \times 100
   \]  
   (2)
   f) Lean mass index (kg/m²)

2) Regional body fat percentages, which included
   a) Percentage of arm region made up of fat (%), which was calculated as
   \[
   \frac{\text{Arm fat mass}}{\text{Arm fat mass} + \text{Arm lean mass} + \text{Arm BMC}} \times 100
   \]  
   (3)
   b) Percentage of leg region made up of fat (%), which was calculated as
   \[
   \frac{\text{Leg fat mass}}{\text{Leg fat mass} + \text{Leg lean mass} + \text{Leg BMC}} \times 100
   \]  
   (4)
   c) Percentage of trunk region made up of fat (%), which was calculated as
   \[
   \frac{\text{Trunk fat mass}}{\text{Trunk fat mass} + \text{Trunk lean mass} + \text{Trunk BMC}} \times 100
   \]  
   (5)
   d) Percentage of android region made up of fat (%), which was calculated as
   \[
   \frac{\text{Android fat mass}}{\text{Android fat mass} + \text{Android lean mass} + \text{Android BMC}} \times 100
   \]  
   (6)
   e) Percentage of gynoid region made up of fat (%), which was calculated as
   \[
   \frac{\text{Gynoid fat mass}}{\text{Gynoid fat mass} + \text{Gynoid lean mass} + \text{Gynoid BMC}} \times 100
   \]  
   (7)

3) Fat mass distribution, which included
   a) Android:gynoid ratio (ie, percentage fat in the android region:percentage of fat in the gynoid region)
   b) Arm fat as a percentage of total body fat (%), which was calculated as
   \[
   \frac{\text{Arm fat mass}}{\text{Total fat mass}} \times 100
   \]  
   (8)
   c) Leg fat as a percentage of total body fat (%), which was calculated as
   \[
   \frac{\text{Leg fat mass}}{\text{Total fat mass}} \times 100
   \]  
   (9)
   d) Trunk fat as a percentage of total body fat (%), which was calculated as
   \[
   \frac{\text{Trunk fat mass}}{\text{Total fat mass}} \times 100
   \]  
   (10)

Eating disorder psychopathology

Eating disorder psychopathology was assessed by using the validated Italian version of the 12th edition of the EDE (21, 22).
The EDE is an investigator-based interview used to assess the frequency of key behavioral and attitudinal aspects of eating disorders over the preceding 4 wk (28 d). The test is scored on a 7-point scale from 0 to 6, with higher scores reflecting greater severity or frequency. The EDE contains 4 subscales (dietary restraint, eating concern, weight concern, and shape concern) and a global score, which provides a detailed and comprehensive profile of the psychopathological features of eating disorder patients (27). The interrater reliability of the EDE has been estimated to be 0.97–0.99 (28), and in this case, the questionnaire was administered by assessors (trained and supervised by RDG, who is an expert in the use of the questionnaire). Assessors had no involvement in the treatment itself.

EDE scores were used to generate 2 primary outcome variables as follows: the change in the severity of eating disorder features and possession of a global EDE score <1 SD above the community mean (ie, <1.74) (29). Finally, data were analyzed for any correlation between body-composition variables and global EDE or shape-concern subscale scores.

**General psychological distress**

General psychological distress was assessed by using a Global Severity Index (GSI) calculated from Brief Symptom Inventory (BSI) scores (validated Italian version) (30, 31). For the assessment, patients score 53 items, which are indicative of 9 symptom dimensions and 3 global indexes of distress, according to their level of concern regarding various symptoms over the preceding week [from 0 (not at all) to 4 (extremely concerned)]. The GSI is the single best indicator of current distress levels, and scores are calculated by summing and dividing by the total number of completed items. Reported norms for adult women indicate a mean (±SD) GSI of 0.35 ± 0.37 for nonpatients (32). The BSI has been shown to have good internal consistency and reliability for the 9 dimensions, with α coefficients that ranged from 0.71 to 0.85 and test-retest reliability coefficients that ranged from 0.68 to 0.91. Good convergent, construct, and predictive validities have been reported (32). In our sample Cronbach’s α was 0.97.

**Statistical analysis**

At baseline, demographic and clinical characteristics of both patients with AN and controls were tested for differences by using the t test for independent samples. The significance of differences in clinical variables from admission to discharge in patients with AN was determined by using the t test for paired data. To evaluate relations between the total and regional body composition and eating disorder psychopathology and general psychological distress, we applied Pearson’s product-moment correlation coefficients as follows: 1) between baseline body-composition variables compared with baseline global EDE and shape-concern subscale scores, and compared with the BSI-GSI score; 2) between end-of-treatment body-composition variables compared with end-of-treatment global EDE and shape-concern subscale scores, and compared with the BSI-GSI score; 3) between the change in body-composition variables from baseline to the end of treatment compared with baseline global EDE and shape-concern subscale scores, and compared with the BSI-GSI score; 4) between the change in body-composition variables from baseline to the end of treatment compared with end-of-treatment global EDE and shape-concern subscale scores, and compared with the BSI-GSI score; 5) between end-of-treatment body-composition variables compared with the change in global EDE and shape-concern subscale scores from baseline to the end of treatment, and compared with the BSI-GSI score; and 6) between the change between baseline and end-of-treatment body-composition variables compared with the change in global EDE and shape-concern subscale scores from baseline to the end of treatment, and compared with the BSI-GSI score.

Significantly correlated variables were inserted into single linear regression models by using global EDE, shape-concern EDE subscale, or BSI-GSI scores as dependent variables, with age, educational level, physical activity level (measured in min/mo according to EDE items), the presence of amenorrhea, and baseline questionnaire scores controlled for, as appropriate.

To adjust P values for multiple comparisons, we applied Holm’s procedure, which is a less-conservative and more-powerful method than Bonferroni’s test (33, 34). P was significant at <0.018. Data were analyzed with SPSS software (SPSS Statistics, version 20.0; IBM).

**RESULTS**

**Baseline clinical characteristics of participants**

Body-composition variables of the 50 patients with AN and 100 controls are shown in Table 2. At baseline, patients with AN had significantly lower weight, BMI, total lean and fat masses, total fat mass percentage, fat and lean mass indexes, and a higher lean mass:fat mass ratio than those of controls. Arm, leg, trunk, and android and gynoid fat mass percentages were also significantly lower in patients with AN than controls, although there was no significant difference between the 2 groups in terms of the android:gynoid ratio. Finally, patients with AN displayed lower percentages of (arm fat ÷ total fat) × 100 and (trunk fat ÷ total fat) × 100 than those of controls but similar percentages of (leg fat ÷ total fat) × 100.

**Effect of treatment**

The treatment was associated with a significant increase in the mean (±SD) weight of patients (11.1 ± 4.1 kg; P < 0.001), which was equivalent to a BMI increase of 4.3 ± 1.6 (P < 0.001). Eating disorder psychopathology and general psychological distress also improved substantially by the program as evidenced by the mean global EDE score, which decreased by 1.7 ± 1.3 (P < 0.001), and the mean BSI-GSI, which fell by 1.1 ± 0.8 (P < 0.001). More than fifty percent of patients (54%; 27 of 50 women) had minimal residual eating disorder psychopathology measured as a global EDE score <1SD above the community mean (ie, <1.74) (28), and 28% of patients (14 of 50 women) showed minimal residual general psychopathology (BSI-GSI <0.35). A comparison of pretreatment and posttreatment body-composition variables in patients with AN indicated significant improvements in each variables from baseline to the end of treatment (Table 3).

Percentages of (arm fat ÷ total fat) × 100 and (leg fat ÷ total fat) × 100 were not significantly different between baseline and the end of treatment, but the percentage of (trunk fat ÷ total fat) × 100
was significantly higher after weight restoration than at baseline (Table 2).

No significant differences were shown between control and posttreatment AN groups in terms of weight, BMI, and total, percentages, or indexes of lean and fat masses. However, after treatment, patients with AN had lower percentages of arm and leg fat masses, higher percentages of trunk, android, and gynoid fat masses, a higher android:gyroid ratio, lower percentages of (arm fat ÷ total fat) × 100 and (leg fat ÷ total fat) × 100, and a higher percentage of (trunk fat ÷ total fat) × 100 than those of the control group (Table 2).

### Body composition, eating disorder psychopathology, and general psychological distress

No significant relation was shown between body-composition variables and global EDE or shape-concern EDE subscale scores, either at baseline, at the end of treatment, or in changes between pre- and posttreatment assessment times.

### TABLE 2

<table>
<thead>
<tr>
<th>Pretreatment AN</th>
<th>Posttreatment AN</th>
<th>Control group</th>
<th>Comparison between pretreatment AN and control groups</th>
<th>Comparison between posttreatment AN and control groups</th>
<th>Comparison between pretreatment and posttreatment AN values</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Difference</td>
<td>$P^2$</td>
<td>Difference</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>39.9 ± 5.1</td>
<td>51.0 ± 4.6</td>
<td>12.5 ± 3.7</td>
<td>&lt;0.001</td>
<td>1.5 ± 0.8</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>15.4 ± 1.6</td>
<td>19.6 ± 0.8</td>
<td>4.4 ± 0.8</td>
<td>&lt;0.001</td>
<td>0.2 ± 0.1</td>
</tr>
<tr>
<td>Total body composition</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total fat mass (kg)</td>
<td>4.1 ± 2.7</td>
<td>12.2 ± 3.2</td>
<td>8.5 ± 0.4</td>
<td>&lt;0.001</td>
<td>0.4 ± 1.0</td>
</tr>
<tr>
<td>Total fat mass (%)</td>
<td>10.0 ± 6.0</td>
<td>23.9 ± 5.8</td>
<td>14.0 ± 2.1</td>
<td>&lt;0.001</td>
<td>0.2 ± 1.8</td>
</tr>
<tr>
<td>Fat mass index</td>
<td>1.6 ± 1.0</td>
<td>4.7 ± 1.2</td>
<td>3.2 ± 0.2</td>
<td>&lt;0.001</td>
<td>0.07 ± 0.4</td>
</tr>
<tr>
<td>Total lean mass (kg)</td>
<td>34.0 ± 4.0</td>
<td>36.8 ± 4.1</td>
<td>3.8 ± 0.6</td>
<td>&lt;0.001</td>
<td>0.9 ± 0.7</td>
</tr>
<tr>
<td>Total lean mass (%)</td>
<td>48.6 ± 0.2</td>
<td>48.7 ± 0.2</td>
<td>0.07 ± 0.1</td>
<td>0.018</td>
<td>0.003 ± 0.06</td>
</tr>
<tr>
<td>Lean mass index</td>
<td>13.1 ± 1.2</td>
<td>14.2 ± 1.1</td>
<td>1.2 ± 0.4</td>
<td>&lt;0.001</td>
<td>0.07 ± 0.3</td>
</tr>
<tr>
<td>Lean mass:fat mass ratio</td>
<td>12.8 ± 7.7</td>
<td>3.3 ± 1.5</td>
<td>−9.5 ± 6.9</td>
<td>&lt;0.001</td>
<td>−0.2 ± 0.7</td>
</tr>
<tr>
<td>Regional body fat (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arm fat mass</td>
<td>7.3 ± 4.9</td>
<td>20.2 ± 7.1</td>
<td>17.6 ± 1.3</td>
<td>&lt;0.001</td>
<td>4.7 ± 0.8</td>
</tr>
<tr>
<td>Leg fat mass</td>
<td>13.4 ± 8.8</td>
<td>27.9 ± 6.7</td>
<td>13.5 ± 5.0</td>
<td>&lt;0.001</td>
<td>3.6 ± 1.8</td>
</tr>
<tr>
<td>Trunk fat mass</td>
<td>8.5 ± 5.3</td>
<td>23.3 ± 6.4</td>
<td>18.8 ± 4.1</td>
<td>&lt;0.001</td>
<td>4.5 ± 2.2</td>
</tr>
<tr>
<td>Android fat mass</td>
<td>8.9 ± 6.2</td>
<td>27.2 ± 7.6</td>
<td>19.4 ± 4.9</td>
<td>&lt;0.001</td>
<td>7.8 ± 2.6</td>
</tr>
<tr>
<td>Gynoid fat mass</td>
<td>18.1 ± 11.2</td>
<td>36.7 ± 6.0</td>
<td>13.6 ± 4.4</td>
<td>&lt;0.001</td>
<td>5.1 ± 1.6</td>
</tr>
<tr>
<td>Fat mass distribution</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Android:gyroid ratio</td>
<td>0.6 ± 0.2</td>
<td>0.7 ± 0.1</td>
<td>0.6 ± 0.1</td>
<td>0.02 ± 0.1</td>
<td>0.508</td>
</tr>
<tr>
<td>Arm fat ÷ total fat (%)</td>
<td>6.5 ± 1.9</td>
<td>7.0 ± 1.6</td>
<td>10.5 ± 4.1</td>
<td>&lt;0.001</td>
<td>4.1 ± 0.3</td>
</tr>
<tr>
<td>Leg fat ÷ total fat (%)</td>
<td>43.7 ± 10.2</td>
<td>42.5 ± 6.3</td>
<td>47.3 ± 4.8</td>
<td>&lt;0.001</td>
<td>3.5 ± 5.4</td>
</tr>
<tr>
<td>Trunk fat ÷ total fat (%)</td>
<td>40.8 ± 7.7</td>
<td>46.1 ± 6.3</td>
<td>35.7 ± 4.3</td>
<td>&lt;0.001</td>
<td>−5.1 ± 3.5</td>
</tr>
</tbody>
</table>

*All values are means ± SDs. Pretreatment assessment occurred in the first week of inpatient admission, and posttreatment assessment occurred in the last week of the 20-wk treatment program, which comprised 13 wk of inpatient therapy followed by 7 wk of partial hospitalization. P values were determined by using the t test for independent samples. P values were determined by using the t test for paired samples. Not significant according to Holm’s procedure (P < 0.018).
the 2 time points. However, significant correlations between the BSI-GSI score and several body-composition variables were detected \((P < 0.05)\). In particular, the baseline BSI-GSI score was significantly correlated with baseline (arm fat \(\div\) total fat) \(\times 100\) \((r = -0.31, P = 0.031)\). There were also significant correlations between the difference between baseline and end-of-treatment BSI-GSI scores and the following end-of-treatment body-composition variables: total fat mass percentage \((r = -0.30, P = 0.036)\), total lean mass \((r = -0.29, P = 0.040)\), lean mass:fat mass ratio \((r = -0.31, P = 0.030)\), arm fat mass percentage \((r = -0.40, P = 0.008)\), trunk fat mass percentage \((r = -0.29, P = 0.044)\), android fat mass percentage \((r = -0.32, P = 0.022)\), and (arm fat \(\div\) total fat) \(\times 100\) percentage \((r = -0.39, P = 0.010)\). Finally, the change in the BSI-GSI score was significantly correlated with both the change in the arm fat mass percentage and change in the android fat mass percentage \((r = -0.36, P = 0.011; r = -0.30, P = 0.032)\), respectively.

Independent linear regression analyses were used to assess the relation between the change in the BSI-GSI score and body-composition variables, with age, education level, presence of amenorrhea, baseline BSI-GSI score, and physical activity level at the end of treatment controlled for. Results indicated that, for each independent variable, only the baseline BSI-GSI score was significantly related with the change in the BSI-GSI score [total fat mass percentage \((\beta = -0.23, t = -1.73, P = 0.092)\), total lean mass \((\beta = 0.19, t = 1.52, P = 0.135)\), ratio of lean mass:fat mass \((\beta = 0.18, t = 1.46, P = 0.153)\), arm fat mass percentage \((\beta = -0.24, t = -1.98, P = 0.055)\), trunk fat mass percentage \((\beta = -0.16, t = -1.72, P = 0.093)\), android fat mass percentage \((\beta = -0.24, t = -1.97, P = 0.056)\), and (arm fat \(\div\) total fat) \(\times 100\) percentage \((\beta = -0.12, t = -0.97, P = 0.338)\), change in arm fat mass percentage \((\beta = 0.22, t = 1.90, P = 0.064)\), and change in android fat mass percentage \((\beta = 0.20, t = 1.77; P = 0.085)\)], which indicated that the change in the BSI-GSI score was significantly predicted only by the severity of psychological distress at baseline.

**DISCUSSION**

**Findings and concordance with previous studies**

The DXA assessment of body composition and fat distribution in 50 adult patients with AN before and after weight restoration and 100 healthy controls matched by age and patients’ post-treatment BMI revealed 3 main findings. The first finding was that, before treatment, patients with AN had lower BMI, lower total lean and fat masses, a lower total fat percentage, lower fat and lean indexes, and a higher lean mass:fat mass ratio than those of controls, but after weight restoration, no significant differences in these variables were shown. This result indicated that patients with AN restored their fat and lean masses completely during the 20 wk of the treatment administered.

The second finding was that there was a marked difference in the body fat distribution between AN and control groups after weight restoration in the former group. Specifically, posttreatment patients with AN had higher fat percentages than did controls in central regions, especially in the android and gynoid regions, but lower fat percentages in peripheral regions. This result supported previous data that, during a relatively rapid weight gain, patients with AN tend to deposit fat mass preferentially in the trunk region (8–11, 13, 35, 36). The android: gynoid ratio we measured also indicated that posttreatment patients with AN had a greater android fat deposition than that of controls. The greater accumulation of body fat in central regions, as opposed to extremities (ie, arm, forearm, thigh, and calf), could have explained the failure, reported in a previous study, of the skinfold technique to quantify the percentage of body fat in patients with AN after weight restoration (37).

The third finding was the absence of any significant relation between eating disorder psychopathology and baseline or end-of-treatment body-composition variables. This result suggested that body shape did not influence the maintenance of the body image disturbance of patients as assessed by the EDE. Moreover, although changes in the BSI-GSI score and some body-composition variables were correlated, the linear regression analysis indicated that the change in the BSI-GSI score was significantly predicted only by the severity of psychological distress at baseline.

**Potential clinical implications**

The absence of any detectable relation between body-composition variables and eating disorder psychopathology should be discussed with patients. This discussion could help patients focus their efforts on addressing the psychological mechanisms that maintain their body-image disturbance rather than attempting to change the shape of their bodies. Likewise, it may be advisable for clinicians to shift the emphasis from changing the body shape and composition of patients with AN during nutritional rehabilitation to focusing more on addressing their eating disorder psychopathology. Moreover, clinicians should pay attention to the psychological distress of patients because this predicts the improvement in general psychopathology during treatment.

Future research should be designed to explore clinical implications of increased truncal fat in women with AN. Indeed, in other populations (eg, the obese) an increase in body fat in the central regions (ie, trunk or android) is associated with metabolic syndrome and is a risk factor for cardiovascular disease, non-alcoholic fatty liver disease, insulin resistance (38), infertility, and ovulation disorders (39). Although one study showed that insulin-resistance onset was associated with a small increase in abdominal fat after a brief period of refeeding in patients with AN (40), no long-term effect data are available.

**Study strengths**

The study has several strengths. First, we assessed 3 levels of body composition (ie, total body composition, regional body fat percentages (which, to our knowledge, were not previously before assessed in patients with AN), and fat mass distribution by using DXA software. DXA, although not yet accepted as a gold-standard technique for body-composition measurement, is known to exhibit a high level of precision in adults with AN, especially in the measurement of body fat (41). Furthermore, this method is rapid, relatively inexpensive, and only relies on a small amount of ionizing radiation. Moreover, the method enables the assessment of regional composition measurements, which provides a distinct advantage compared with approaches such as bioelectrical impedance or hydrodensitometry (42). Second, we assessed patients with AN both before and after body weight normalization, which
enabled us to compare the 2 sets of data. Third, the EDE, which is the gold standard instrument for the assessment of eating disorder psychopathology, and BSI, which is widely recognized in clinical and research settings as a measure of psychological distress, were used. Fourth, our patients with AN were compared with a large sample of healthy controls matched by age and BMI achieved by patients with AN after treatment.

**Study limitations**

Our study did have some limitations. First and foremost, data were obtained in a single inpatient unit by applying one treatment program, which meant that external validation is required. Furthermore, our results need to be interpreted with caution because they may not apply to patients with AN treated in outpatient settings, where a slower rate of weight regain could result in a different regional fat accumulation. However, one outpatient study has already shown a central accumulation of body fat in these subjects, even though weight regain was slower (9). It should also be noted that the less-intensive treatment offered in outpatient settings may have a less pronounced effect in terms of mitigating concerns and psychological distress of patients associated with central body fat accumulation.

Second, DXA assessment may underestimate lean mass in underweight patients with AN because, as showed by Haas et al (43), patients who have a modest reduction in lean mass can be severely protein depleted. This effect is because starvation-induced changes in body composition may be associated with an increase in total body water, which can, in turn, mask low total body protein when a 3-compartment model, as in DXA, is used. Hence, direct methods may be more reliable means of assessing lean mass in underweight patients.

Third, without a long-term follow-up, we were unable to clarify whether lipodystrophy characterized by the preferential accumulation of fat in the central region is a transitory phenomenon, as suggested by a study in 16 patients with AN who maintained normal body weight 1 year after hospital discharge (13) or whether it persists long-term after weight restoration. Hence, future studies should also take into account the effect of time on body composition by serially assessing patients with AN and research settings as a measure of psychological distress, were used. Fourth, our patients with AN were compared with a large sample of healthy controls matched by age and BMI achieved by patients with AN after treatment.

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