

Bone Density of Women Who Have Recovered from Anorexia Nervosa

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Accepted 3 December 1998

Abstract: Objective: To examine bone density in 19 women who had previously experienced classical anorexia nervosa from which they had been fully recovered for a mean of 21 years (current characteristics: median age 40.2 years; Eating Attitudes Test [EAT] score 2; body mass index [BMI] 21.1; average 1.8 offspring). **Method:** Probands were compared, blindly, in respect of bone density, with 13 control subjects matched for age and sex and with no history of eating disorders. Dual energy X-ray absorptiometry (DXA) was used to evaluate the bone mineral density (BMD) of the lumbar spine and the head of the femur. **Results:** Femur BMD was still significantly less among ex-anorectic sufferers. Two subjects had experienced pathological fractures while anorectic, both having been strenuous exercisers. None appeared to have suffered post illness fractures. BMD at follow-up did not relate to the severity or chronicity of previous anorexia nervosa. **Discussion:** Full clinical recovery from anorexia nervosa does not quite confer full establishment of normal bone density. However, pathological fractures are not a feature thereafter, within middle life. © 2000 by John Wiley & Sons, Inc. *Int J Eat Disord* 28: 107–112, 2000.

Key words: bone density; anorexia nervosa; pathological fractures

INTRODUCTION

Skeletal abnormalities have long been described in anorexia nervosa. In 1969, Crisp reported the finding that patients had significantly narrower pelvises than controls taking

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into account height. He attributed this to illness-related estrogen deficiency arising in late puberty. In 1972, Toms and Crisp reported on stunted growth in a young male anorectic, onset aged 12/13 years. Growth recommenced following nutritional recovery. Subsequently, Lacey, Crisp, Hart, and Kirkwood (1979) described delayed bone development in a series of anorectic adolescents, using X-rays of carpal bones to compare radiological with chronological age. Ayers, Gidwani, Schmidt, and Gross (1984) used bone histiometry to confirm the presence of developmental delay and also found evidence of osteopenia. A clinical study by Szmukler, Brown, Parsons, and Darby (1985) of patients with chronic anorexia nervosa found a high incidence of osteoporosis and vertebral fractures in this group. Reduced bone density is therefore of clinical importance; young anorectics may suffer irreversible developmental and growth retardation and older chronic anorectics are at higher risk of pathological fractures.

Most studies have examined clinically ill and underweight anorectics (Salisbury & Mitchell, 1991), so it is clearly important to examine weight-restored anorectics in order to evaluate the possible long-term association between anorexia nervosa and osteoporosis. Conflicting data exist about weight gain and changes in bone mineral density (BMD). Thus, Rigotti, Neer, Skates, Herzog, and Nussbaum (1991) prospectively studied a group of anorectics during weight restoration. They found that BMD did not recover with short-term weight restoration, whereas Orphanidou, McCargar, Birmingham, and Belzberg (1997) found substantial increases in BMD with short-term weight restoration. Cross-sectional studies are also inconsistent. Treasure & Russell (1987) compared clinical anorectics with a group of recovered anorectics and found that bone density was normal in patients who had recovered. However, criteria and duration of recovery were not adequately described in this study. Conversely, both Iketani, Kiriike, Nakanishi, and Nakasuji (1995) and Kooh, Noriega, Leslie, Muller, and Harrison (1996) observed that weight restoration may not be associated with normalization of bone density. Therefore, uncertainty exists about restoration of bone density with complete and sustained clinical recovery.

The etiology of osteoporosis in anorexia nervosa is also unresolved. Osteoporosis has been related to duration of amenorrhea by Lacey et al. (1979), Szmukler et al. (1985), Ayers et al. (1984), Kiriike et al. (1992), and Andersen, Woodward, and LaFrance (1995). However, this relationship was not confirmed by Bachrach, Guido, Katzman, Litt, and Marcus (1990). Salisbury and Mitchell's (1991) review concludes that osteoporosis is probably related to duration of amenorrhea, which suggests a causal role for low estrogen level.

Early age of onset of anorexia nervosa has been consistently found to predict osteoporosis (Salisbury & Mitchell, 1991; Seeman, Szmukler, Formica, Tsalamandris, & Mestrovic, 1992; Bachrach et al., 1990; Ayers et al., 1984). Other etiological controversies such as the role of high cortisol levels, exercise habits, and dietary abnormalities have been reviewed by Salisbury and Mitchell (1991).

This study was intended to answer the following questions: Does BMD return to normal in anorectic patients who are fully recovered? What is the long-term risk of pathological fractures in recovered patients? In this group of subjects, is low bone density associated with duration of illness, age of onset, or lowest weight ever?

METHOD

This project forms part of an ongoing long-term follow-up of female anorectic patients first seen by this department between 1968 and 1972. These 105 patients have now been

followed up for 20 years (Crisp, Callender, Halek, & Hsu, 1992; Hsu, Crisp, & Callender, 1992). Thirty women, recovered beyond clinical dispute (and also according to the general outcome categories devised by Morgan & Russell, 1975), were contacted and 19 agreed to participate in this study. Thirteen control subjects were recruited from the staff of the medical school: these were women of a similar age who, it was concluded after questioning, had never suffered from anorexia nervosa.

Dual-energy X-ray absorptiometry (DXA) was used to evaluate, blindly, the BMD of the lumbar spine and head of femur of subjects and controls. Age, weight, and height were recorded for both subjects and controls. Eating habits of subjects were measured by the Eating Attitudes Test (EAT; Garner & Garfinkel, 1979). Detailed clinical information was obtained from subjects using a semistructured interview, which was supplemented by referring to case notes and to a computerized clinical database where necessary. This clinical information included age of onset of illness, lowest weight ever, duration of low weight (below 85% of mean matched population weight), duration of amenorrhea, and history of exercise, fractures, and oral contraceptive use.

BMD, body mass index (BMI), and age of subjects and controls were compared using the Wilcoxon two-sample test. In the group of recovered anorectics, the association of BMD with age of onset, lowest weight ever, and duration of illness was evaluated using Pearson's correlation coefficient. Data are presented as median (interquartile range).

RESULTS

Table 1 indicates some salient clinical characteristics of the group of recovered anorectic patients. In particular, these subjects had been normal in weight for a median of 21 years. They had, since recovery, produced between them 35 live children (mean 1.86, range 0–4). Their median current EAT score was 2, confirming that this group was not merely weight restored but also in full remission from anorexia nervosa.

Table 2 compares subjects and controls with regard to current age, BMI, and BMD. Subjects and controls were of comparable age, the median age of subjects being 3.1 years older than the median age of controls ($p = .774$). They were also of comparable weight, with median BMI values of 21.1 for subjects and 22.4 for controls ($p = .065$). The median BMD was lower in subjects than in controls in both spine and femur measurements: lumbar spine BMD was 1.245 g/cm² in subjects and 1.303 g/cm² in controls; femur BMD was 0.922 g/cm² in subjects and 1.073 g/cm² in controls. This difference was not statistically different for the spine measurement ($p = .374$), but it was for the femur measurement ($p = .004$).

Within the recovered anorectic group, BMD did not correlate with duration of illness either as measured by duration of weight below 85% of expected weight or by duration of amenorrhea. BMD also did not correlate with age of onset. BMD had a weak inverse correlation with lowest ever weight of subjects (Table 3).

Table 1. Clinical data for subjects

	Median	Interquartile Range
Age of onset (years)	16	14–17
Duration of illness at presentation (years)	3	2–3
Lowest weight (kg)	35	31–38
Time since recovery (years)	21	14–23

Table 2. Comparative data for subjects and controls

	Subjects (N = 19)	Controls (N = 13)	Difference	p Value*
Median age (years)	40.2	37.1	3.1	.774
IQR age (years)	34.4–42.6	35.7–42.8	—	
Median current BMI	21.2 m/kg ²	22.4 m/kg ²	1.3 m/kg ²	.065
IQR current BMI	20.1–22.4 m/kg ²	21.4–27.7 m/kg ²	—	
Median bone density spine	1.245 g/cm ²	1.303 g/cm ²	0.058 g/cm ²	.347
IQR bone density spine	1.110–1.275 g/cm ²	1.167–1.418 g/cm ²	—	
Median bone density femur	0.922 g/cm ²	1.073 g/cm ²	0.151 g/cm ²	.004
IQR bone density femur	0.839–0.993 g/cm ²	0.989–1.126 g/cm ²	—	
Median current EAT score	2	Not available	—	
IQR current EAT score	14	Not available	—	

Note: IQR = interquartile range; BMI = body mass index; EAT = Eating Attitudes Test.

*Data are not normally distributed. *p* values calculated using Wilcoxon two-sample test.

Three of the 19 subjects had a history of fractures, and the clinical histories of these individuals were examined in more detail. One was excluded as her fractures followed severe trauma and were unlikely to have been related to osteoporosis. The other 2, referred to here as Subject A and Subject B, both had a history of two pathological fractures while underweight. Subject A had a history of chronic illness with amenorrhea and low weight between the ages of 15 and 25. Her lowest weight ever had been 30.4 kg. She had undertaken strenuous exercise while she was underweight, and currently undertakes regular non-weight-bearing exercise. Her current BMI is 21.1. Her current lumbar spine BMD is 1.257 g/cm² (108% of population norm) and her femoral BMD is 1.021 g/cm² (111% of population norm). Subject B has a relatively brief history of anorexia nervosa with 2 years of amenorrhea and 18 months of low weight. Her lowest weight ever was 40 kg. She exercised strenuously when underweight but does not do any regular exercise currently. Her current BMI is 30.4. Her current lumbar spine BMD is 0.966 g/cm² (82% of population norm) and her femoral BMD is 0.731 g/cm² (71% of population norm): Subject B is still at high risk of osteoporosis and pathological fractures.

CONCLUSION

This study set out to examine the BMD of long-term recovered anorexics. In our subjects, BMD did not correlate with duration of illness or age of onset. The weak negative

Table 3. Correlation of clinical variables with bone mineral density

	Correlation Coefficient	95% Confidence Interval
Duration of amenorrhea		
Spine	$r = 0.08$	$-0.39 < r < 0.52$
Femur	$r = 0.19$	$-0.29 < r < 0.59$
Duration of low weight		
Spine	$r = -0.01$	$-0.47 < r < 0.46$
Femur	$r = 0.27$	$-0.22 < r < 0.66$
Age of onset		
Spine	$r = 0.06$	$-0.40 < r < 0.50$
Femur	$r = 0.00$	$-0.45 < r < 0.45$
Lowest weight ever		
Spine	$r = 0.20$	$-0.61 < r < 0.29$
Femur	$r = 0.49$	$-0.78 < r < -0.03$

correlation between lowest weight ever and femoral BMD after recovery is implausible and this is likely to be a chance finding.

The results indicate that even in individuals who have been clinically well for many years (a median of 21 years since recovery), BMD does not fully return to normal. This is clearly the case with regard to femur BMD, which is significantly less in the recovered anorectic group. Lumbar spine BMD is lower in this group but not at a statistically significant level. This might be a chance finding and a reflection of the relatively small sample size: a larger study might find lower BMD in both the lumbar spine and in the femur of recovered anorectics.

Another possibility is that there is a real difference in the vulnerability to mineral loss, or failure of remineralization, between vertebral and appendicular bone in anorectics. If this is true, our findings are not in keeping with those of Mazess, Barden, and Ohlrich (1990), Davies et al. (1990), Bachrach et al. (1990), Herzog et al. (1993), and Siemers, Chakmakjian, and Lench (1996). They found that vertebral bone was affected more severely than that of the appendicular skeleton and that spinal osteoporosis correlated more closely with the severity of anorexia nervosa. With the exception of Herzog et al. (1993), these earlier reports were done with underweight anorectics and are not strictly comparable with the present study. To answer this question, one would ideally carry out serial prospective DXA studies on a group of underweight anorectics, following them up during treatment and after a period of sustained recovery.

Estrogen replacement or calcium supplementation is recommended for osteoporosis in anorectics who are chronically underweight (Serpell & Treasure, 1997). However, our results emphasize the importance of treatment of the underlying condition by restoration of normal body weight, to ensure normal skeletal development.

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