

Sarcopenia



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Sarcopenic obesity worsens bone strength: hip strength analysis in post-menopausal women A. Moretti¹, M.T. Giamattei¹, A. de Sire¹, G. Cannaviello¹, F. Gimigliano², R. Gimigliano¹, G. Iolascon¹

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introduction

Increasing knowledge suggests that both osteoporosis and sarcopenia originate from several common denominators.¹ Both fat and muscle tissue provide systemic and local stimuli, including mechanical strain that modulate bone mass and strength.^{2,3} Low bone mass, sarcopenia and obesity have common pathogenetic factors, such as chronic low-grade inflammation, inadequate nutrition, endocrine disorders, low level of physical activity, and neuromuscular disregulation.¹

Estrogens have a protective role for muscle and bone, and estrogens depletion after menopause may explain the natural increase in adipogenesis, weight gain, and accelerated loss of muscle and bone mass.⁴ Adipose tissue, and particularly visceral fat, generates many adipokines, such as leptin, whose receptors are expressed by osteoblasts and myocytes, that could maintain a low-grade inflammatory milieu, contributing to worsening of several clinical conditions, including obesity, osteoporosis and sarcopenia.^{5,6,7} For years, it has been hypothesized that obesity exerted protection against bone loss after menopause,⁸ but recently Nielson et al. reported that osteoporotic fractures occur at higher levels of BMD in obese subjects.⁹

Sarcopenic obesity can be observed clinically as the result of various factors including menopause.¹⁰

Probably, obesity increases the percentage of intramuscular fat mass infiltration, lowering muscle quality and physical performance, with increased risk of falls and fractures.^{1,11} Moreover, the redistribution of adipose tissue might play a role to determine bone strength, particularly at femoral neck.

The aim of our study is to investigate the influence of sarcopenic obesity on hip bone strength indices: Femoral Strength Index (FSI), Cross-Sectional Moment of Inertia (CSMI), Cross-Sectional Area (CSA), Section Modulus (Z) and Buckling Ratio (BR).

There is no conflict of interest that could be perceived as prejudicing the impartiality of the research project.

Obesity

Chronic low-grade inflammation Inadequate nutrition Endocrine disorders Low level of physical activity Neuromuscular disregulation

Low Bone Mass

methods



In this retrospective case-control study, participants were recruited among patients that referred to a Physical Medicine and Rehabilitation outpatients' department for management of osteoporosis, from January 2011 to December 2013. Inclusion criteria were: post-menopausal women aged 50 or older; BMI \geq 30 kg/m². Women who had current or previous HRT (Hormone Replacement Therapy), secondary osteoporosis or pathological fractures were excluded from the assessment. The patients' total and regional body composition indices (ASM - appendicular skeletal muscle mass; FM – total fat mass), bone mineral density (BMD) at total body (total-body less head; TBLH), at the lumbar spine (LS) (anteroposterior projection at L1–L4) and at femoral neck (FN), were measured with the Dual-Energy X-Ray Absorptiometry method (i-DXA-GE Healthcare densitometer). The World Health Organization classification system was applied to define osteoporosis (T-score <-2.5), osteopenia (-2.5<T score<-1), and obesity (BMI – body mass index > 30 kg/m²). For the definition of sarcopenia we used the ASM, calculated by adding lean mass of the upper and lower limbs expressed in kg, adjusted for height (m) and total fat mass (kg) (residuals).¹² Hip Structural Analysis (HSA) was performed from hip DXA images to measure FSI, CSMI, CSA, Z and BR. For each patient we reported anthropometric characteristics, BMI, ASM (residuals), total-body BMD and T-score, femoral neck BMD and T-score, lumbar spine BMD and T-score, and Hip Structural Analysis parameters.



results

We evaluated 127 women, mean aged 63.50 years ± 8.69 Standard Deviation (SD) (min. 50 years; max. 84 years) with a mean BMI ± SD of 34.27 ± 4.01 kg/m².

All the included patients were classified into 2 groups: 45 sarcopenic obeses (35.43%), mean aged 62.69 years \pm 8.50 SD, with a mean BMI \pm SD of 35.36 \pm 4.94 kg/m², and 82 non sarcopenic obeses (64.57%), mean aged 63.96 years \pm 8.81 SD, with a mean BMI \pm SD of 33.68 \pm 3.28 kg/m².

In the sarcopenic obeses group, HSA from hip DXA scans demonstrated a FSI of 1.17 ± 0.33 SD, a mean CSMI of 9586 ± 2417 mm⁴, a mean CSA of 133 ± 23 mm², a mean Z of 554 ± 114 mm³ and a mean BR of 9.11 ± 4.23 SD.

The hip bone strength indices in the non sarcopenic obeses were: a mean FSI of 1.30 ± 0.30 SD, a mean CSMI of 9866 ± 1970 mm⁴ and a mean CSA of 135 ± 21 mm², a mean Z of 571 ± 102 mm³ and a mean BR of 7.11 ± 2.67 SD. DXA scans showed a mean FN BMD of 0.854 ± 0.13 g/cm² and a mean LS BMD of 1.034 ± 0.18 g/cm² in the sarcopenic obeses; a mean FN BMD of 0.860 ± 0.13 g/cm² and a mean LS BMD of 1.080 ± 0.16 g/cm² in the non sarcopenic obeses. Among sarcopenic obeses women, the 33.33% had at least one vertebral fragility fracture and the 40% were osteoporotic. On the other hand the 28.05% of non sarcopenic obeses had vertebral fragility fractures and 36.59% received a diagnosis of osteoporosis.

	Study population (n=127)	Sarcopenic obeses (n=45)	Non-sarcopenic obeses (n=82)		
Age (years)	63.50±8.69	62.69±8.50	63.96±8.81		
Weight (kg)	84.39±10.18	88.47±11.94	82.16 <u>+</u> 8.32		
Height (m)	1.57±0.06	1.58±0.06	1.56±0.06		
BMI (kg/m²)	34.27±4.01	35.36±4.94	33.68 <u>+</u> 3.28		
ALM (kg)	18.27±2.29	17.29±2.25	18.82±2.13		
ASMMI (kg/m²)	7.42±0.86	6.90±0.86	7.70±0.72		
Total FM (kg)	40.17±7.88	45.19 <u>+</u> 8.58	37.41±5.91		
ASM Residuals*	-0.93±3.24	-3.26±1.28	0.35±1.49		
*Appendicular lean mass adjusted for height and body fat mass (residuals). Note: continuous data are expressed as mean + SD					

	Study population (n=127)	Sarcopenic obeses (n=45)	Non-sarcopenic obeses (n=82)		
TBLH BMD (g/cm²)	1.032±0.11	1.028±0.12	1.034±0.11		
TBLH T-score	-0.45±1.10	-0.50±1.17	-0.43±1.06		
FN BMD (g/cm²)	0.858±0.13	0.854 <u>+</u> 0.13	0.860±0.13		
FN T-score	-1.02±1.09	-1.05±1.08	-1.00±1.10		
LS BMD (g/cm²)	1.063±0.17	1.034±0.18	1.080±0.16		
LS T-score	-0.96±1.38	-1.22±1.52	-0.81±1.28		
Vertebral fragility fractures (n)(%)	38 (29.92%)	15 (33.33%)	23 (28.05%)		
Osteoporotic patients (n)(%)	48 (37.80%)	18 (40.00%)	30 (36.59%)		
Note: continuous data are expressed as mean <u>+</u> SD					

	Study population (n=127)	Sarcopenic obeses (n=45)	Non-sarcopenic obeses (n=82)		
FSI	1.25±0.31	1.17±0.33	1.30±0.30		
CSMI (mm ⁴)	9766.90±2133.71	9586.02 <u>+</u> 2416.68	9866.16±1970.11		
CSA (mm²)	134.25 <u>+</u> 21.64	133.47±23.13	134.68±20.91		
Z (mm³)	564.76±105.96	553.87±113.57	570.73±101.77		
BR	7.82±3.43	9.11±4.23	7.11±2.67		
Note: continuous data are expressed as mean <u>+</u> SD					

conclusions

In our cohort of post-menopausal women, sarcopenic obeses had worse bone quality and strength than non-sarcopenic obeses. This finding is supported by an increase of buckling ratio, which is the ratio of the outer radius to the cortical thickness, that represents a mean for estimating stability of the cortex in thin-walled regions subjected to bending. The buckling ratio uniquely reflects the transition from strength homeostasis to skeletal fragility. In our population, sarcopenic obeses women with higher buckling ratio showed an higher frequency of vertebral fragility fractures, probably linked to a widespread impaired bone strength.

Our data furtherly supported the hypothesis that excess adiposity (over-weight/obesity) should be an integral component of the diagnosis of both sarcopenia and osteoporosis.^{13,14} HSA should be included in sarcopenic obesity assessment, representing an important evaluation tool to define the close relationship among fat, muscle, and bone that influence each other on a biomolecular as well as on tissue level.

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