◎原 著

Glucocorticoid-induced cortical bone porosity in postmenopausal patients with asthma

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Abstract : In a previous study, we demonstrated that chronic administration of systemic glucocorticoids decreases cortical bone mineral density (BMD) and induces development of pathologic fractures in asthmatic patients. To investigate cortical bone porosity due to glucocorticoids, we studied cortical bone volume, BMD, bone strength, and fractures in patients with asthma in this report.

A total of 82 postmenopausal asthmatic patients were enrolled in the study. Vertebral fractures were diagnosed via plain spinal radiograms. Peripheral quantitative computed tomography (pQCT) was used to measure cortical BMD, relative cortical volume, and Strength Strain Index (SSI). Multiple regression analysis, Student's t test, and other statistical analyses were performed.

Patients with systemic glucocorticoids therapy had lower cortical BMD, relative cortical volume, SSI, and more number of vertebral fractures than patients without it. Lifetime cumulative dose of glucocorticoids was related to cortical BMD, relative cortical volume, SSI, and the number of vertebral fractures. The cortical volume-density relationship appeared to remain constant regardless of systemic glucocorticoid administration. The number of vertebral fractures correlated highly with cortical BMD, relative cortical volume, and SSI at the radius.

In conclusion, systemic glucocorticoid administration decreases cortical bone density, cortical bone volume, and bone strength. Glucocorticoid administration appears to be responsible for the process of cortical bone porosity at both endosteal and intracortical sites. Given that both cortical bone density and volume provide bone strength, cortical bone porosity was seen to contribute to glucocorticoid-induced bone strength loss and fractures.

Key words : glucocorticoid, cortical bone, pQCT, osteoporosis

Introduction

Recently, techniques for measuring bone mineral density (BMD) have been developed. Peripheral quantitative computed tomography (pQCT) measures three-dimensional density, and separately determines both cortical bone density and trabecular bone density with a high level of precision (0.8 to 1.6% coefficient of variation)[1-3]. In addition, pQCT is able to noninvasively estimate bone strength based on the theory on the stability of mechanical structures against bending or torsion[4, 5]. pQCT on the radius is also known to provide an accurate assessment of the general condition of bone[6-8]. Prior to the development of pQCT, axial quantitative computed tomography (QCT) was used, but this was less precise[9] and the cortical shell of vertebral bodies was too thin to be measured accurately[10, 11]. Other standard densitometric methods, such as dual energy X-ray absorptiometry (DXA) gave little or no information about bone quality or distribu-tion[9].

Trabecular bone and cortical bone differ in their remodeling characteristics[12, 13], and structure[12]. Cortical bone is closely related to bone strength and stiffness[7, 14]. Although the strength and stiffness of bone are positively correlated with cortical bone density[7, 14], cortical bone volume is also known to influence bone strength[4, 5, 7, 11, 15].

Using pQCT, previous studies have reported that aging is associated with a reduction in cortical bone volume as well as a reduction in cortical bone density[10, 12-16]. Fujii et al. demonstrated that a fixed ratio exists between the radius cortical volume and density in healthy adult humans regardless of either age or sex, suggesting that cortical bone porosity causes similar and simultaneous decreases in cortical bone volume and density during the aging process[11]. In secondary hyperparathyoidism, similar findings were observed in uremic patients on maintenance hemodialysis[17].

Chronic use of systemic glucocorticoids (GC) is known to lead to progressive bone loss and the development of pathologic fractures in a correlating manner with the cumulative dose of GC[18-21]. Some patients with severe asthma require prolonged oral systemic GC therapy despite the development of inhaled steroid therapy, and these patients are at a high risk for developing skeletal complications due to GC therapy[18-24]. Recently, we demonstrated using pQCT that systemic GC administration decreases cortical bone density and trabecular bone density in asthmatic patients and leads to the development of pathologic fractures[8]. This effect is most pronounced in older patients[8].

In GC-induced bone loss, however, bone strength, cortical volume, and the relationship between cortical volume and density concerning cortical bone porosity have to date never been studied. In the present study, we used pQCT to measure changes in bone strength and cortical bone volume in addition to cortical and trabecular BMD in patients with asthma undergoing systemic glucocorticoid therapy.

Materials and Methods

SUBJECTS

Data from 82 outpatients with asthma were selected for multiple regression analysis in this study. All 82 patients were naturally postmenopausal women who had gone through menopause at least 10 years prior to the start of the study (mean age: 70.6±5.4 years, and mean years since menopause: 21.8 ±6.2) (Table 1). All patients had taken

inhaled GC (range: 200 to 800 μg/day of beclomethasone dipropionate; mean: 338 μg/day) over a period of 2 to 8 years (mean: 4.6 years). Of the 82 patients with asthma, 60 were assigned to one of two groups for a between groups comparison (Table 2). In the GC(-) group, 24 patients without systemic GC therapy received less than a 0.5 g cumulative dose of prednisolone. In the GC(+) group, 36 patients with systemic GC therapy received more than a 10 g cumulative dose of prednisolone (mean: equivalent of 24.1 g of prednisolone) over a prolonged period (mean: 9.5 years). There were no significant differences in age, age at menopause, years since menopause (YSM), height, weight, or body mass Index (BMI) between groups (Table 2). Patients who had taken medication that affects bone metabolism or patients with medical conditions that affect bone metabolism were excluded from the study. No patient had undergone treatment for osteoporosis, such as hormone replacement therapy. No patient had alcohol dependency, and no patient was a smoker. A history was taken, and a physical examination was performed on all patients. Duration, daily dose, and lifetime cumulative dose of GC were calculated from medical records and a patient's personal records. The dose of GC is expressed in equivalent grams of prednisolone.

Table 1. Patient Characteristics.

	All Patients (n=82)
Age (years)	70.6±5.4
Age at menopause (years)	48.8±4.6
Years since menopause (years)	21.8±6.2
Height (cm)	148.8±5.8
Weight (kg)	50.6±7.2
BMI (kg/m^2)	22.9±2.9
Total BMD (mg/cm^3)	281.0±61.3
Trabecular BMD (mg/cm^3)	116.9±32.9
Cortical BMD (mg/cm^3)	1065.6±60.7
Relative cortical volume	0.438±0.074
Strength Strain Index (mm^3)	179.3±49.5
Number of vertebral fractures	1.20±1.89
Cumulative dose of prednisolone (g)	15.1±24.8

Values are presented as the mean±SD.
BMI: body mass index; BMD: bone mineral density

METHODS

All patients were evaluated for the presence of vertebral deformities on lateral lumbar and thoracic radiographs. A vertebral fracture was defined as an altered morphology and a decrease in vertebral height of approximately 25% or more at the anterior, middle, or posterior aspect of the vertebral body.

pQCT was performed on the non-dominant radius using a Stratec XCT 960 (Nishimoto,

Table 2. Patient Characteristics Divided by Cumulative Dose of Glucocorticoid Use.

	GC(−) group [a] (n=24)	GC(+) group [b] (n=36)	t test
Age (years)	71.0±4.6	69.3±5.4	N.S.
Age at menopause (years)	49.2±3.0	47.5±5.5	N.S.
Years since menopause (years)	21.8±6.0	21.7±6.6	N.S.
Height (cm)	149.1±6.5	148.3±5.2	N.S.
Weight (kg)	52.7±7.9	49.9±5.8	N.S.
BMI (kg/m^2)	23.7±3.2	22.7±2.6	N.S.
Cumulative dose of prednisolone (g)	0.08±0.19	24.1±25.9	$p< 0.01$

Values are presented as the mean±SD.
GC: glucocorticoids; BMI: body mass index; BMD: bone mineral density; N.S.: not significant
a: patients without systemic GC therapy who received less than a 0.5g cumulative dose of prednisolone
b: patients with sytemic GC therapy who received more than a 10g cumulative dose of predonisolone

Tokyo, Japan), as described previously[8, 25]. Briefly, the ultradistal radial 4% site (at 4% of the ulnar length proximal to the distal end of the radius) was used to calculate trabecular BMD and total BMD, while the mid-radial 20% site was used to calculate cortical BMD. Constant threshold levels were used for all subjects (0.5 mg/cm³ for total bone and 0.93 mg/cm³ for cortical bone).

Total and cortical area were estimated based on voxel number at the mid-radial 20% site region, and relative cortical volume, defined as cortical volume divided by the total bone volume, was calculated by dividing cortical area with total area, as described previously[11, 26].

Determination of bone strength was based on calculation of the cross-sectional moment of inertia divided by the maximum distance of any voxel from the center of gravity. This section modulus represents the geometrical properties of the bone and is directly proportional to the maximum stress in the bone. The material properties were not measured directly but estimated by multiplying the section modulus by the normalized cortical density (to a normal physiological density of 1200 mg/cm³). By combining these geometrical and material properties, we calculated the Strength Strain Index (SSI) values[4, 5, 16]. This index was calculated for the mid-radial 20% site region, and the default threshold setting of 0.7 mg/cm³ was used in relation to the amount of cortical bone present, as described previously[4, 5, 16].

Statistical analysis

Student's t test, multiple regression analysis, and other statistical analyses were performed using the software package, StatView (Abacus Concepts, Berkeley, CA, USA). A p value < 0.05 was considered to be significant.

Results

In all 82 patients, multiple regression analysis was performed for total BMD, trabecular BMD, cortical BMD, relative cortical volume, SSI, and the number of vertebral fractures to determine the predictive value of the following variables : YSM, lifetime cumulative dose of GC, and BMI. Multiple regression analysis demonstrated significant relationships ($p< 0.0001$) between each of these variables and total, trabecular, and cortical BMD, relative cortical volume, SSI, and the number of vertebral fractures. YSM was significantly correlated with cortical BMD, relative cortical volume, and SSI (Table 3, 4). Lifetime cumulative dose of GC was significantly correlated with total BMD, trabecular BMD, cortical BMD, relative cortical volume, SSI, and the number of vertebral fractures (Table 3, 4, 5). BMI was significantly correlated with total BMD, trabecular BMD, and relative cortical volume (Table 3, 4).

Spearman's rank correlation test was performed on all 82 patients, and the number of vertebral fractures was seen to be significantly correlated ($p < 0.0001$) with cortical BMD, relative cortical volume, and SSI at the radius.

Significant differences ($p < 0.01$) were observed between the GC(+) group and the GC(-) group in total BMD, trabecular BMD, cortical BMD, relative cortical volume, SSI, and the number of vertebral fractures (Table 6).

A significant correlation between relative cortical volume and cortical BMD showing a rectilinear relationship was observed in both

Table 3. Prediction of Bone Mineral Density by Multiple Regression Analysis.

Predictor Variable	Total BMD (R^2= 0.383)			Trabecular BMD (R^2= 0.313)			Cortical BMD (R^2= 0.554)		
	Regression Coefficient	SE	p-value	Regression Coefficient	SE	p-value	Regression Coefficient	SE	p-value
YSM	-1.579	0.891	0.0803	-0.31	0.504	0.5405	-3.190	0.754	< 0.0001
Cumulative PSL	-0.001	0.0002	0.0007	-0.0005	0.0002	0.0003	-0.001	0.0002	< 0.0001
BMI	6.184	1.610	<0.0001	3.765	1.095	0.0009	1.508	1.632	0.3584

BMD: bone mineral density; SE: standard error;

YSM: Years since menopause; BMI: body mass index; PSL; prednisolone

Table 4. Prediction of pQCT Parameters by Multiple Regression Analysis.

Predictor Variable	SSI (R^2= 0.263)			Relative Cortical Volume (R^2= 0.486)		
	Regression Coefficient	SE	p-value	Regression Coefficient	SE	p-value
YSM	-2.530	0.790	0.0020	-0.351	0.099	0.0007
Cumulative PSL	-0.001	0.0002	0.0125	-0.0001	0.00003	< 0.0001
BMI	3.251	1.709	0.0609	0.699	0.214	0.0017

SE: standard error; YSM: Years since menopause;

BMI: body mass index; PSL; prednisolone; SSI: Strength Strain Index

the GC(-) group ($p < 0.0001$, $r^2 = 0.772$) and the GC(+) group ($p < 0.0001$, $r^2 = 0.727$) (Figure 1).

No significant differences were observed in the two cortical volume-density slopes of patients with or without systemic GC therapy.

Discussion

In women, there is an irreversible and substantial loss of bone consequent to the lack of estrogen in the 10 year period following menopause[27]. Previous reports have detected perimenopausal bone loss in both trabecular and cortical bone using pQCT[10]. In a previous report, we demonstrated that systemic GC administration decreases the density of both trabecular and cortical bone, and that this effect is most pronounced in older patients[8]. In that report, however, although gender was corrected for potential confounding with multiple regression, we gave little consideration to the influence of menopause[8]. In the present study, to exclude the influence of perimenopausal bone loss consequent to a lack of estrogen, we examined female patients who had gone through menopause more than ten years prior to the study.

Table 5. Prediction of Vertebral fractures by Multiple Regression Analysis.

Predictor Variable	Vertebral fractures (R^2= 0.491)		
	Regression Coefficient	SE	p-value
YSM	0.034	0.025	0.1795
Cumulative PSL	0.00005	0.000006	< 0.0001
BMI	-0.059	0.054	0.2786

SE: standard error; YSM: Years since menopause;

BMI: body mass index; PSL; prednisolone

In the present study, we have demonstrated that systemic GC administration decreases the density of both trabecular and cortical

Table 6. Comparison Within Patient Sub-groups.

	GC(−) group [a] (n=24)	GC(+) group [b] (n=36)	t test
Total BMD (mg/cm^3)	317.7±68.6	255.7±40.3	p< 0.01
Trabecular BMD (mg/cm^3)	142.2±27.1	98.1±21.0	p< 0.01
Cortical BMD (mg/cm^3)	1096.5±44.1	1048.5±50.0	p< 0.01
Relative cortical volume	0.486±0.061	0.407±0.061	p< 0.01
Strength Strain Index (mm^3)	207.2±40.8	162.9±52.4	p< 0.01
Number of vertebral fractures	0.13±0.61	1.61±1.57	p< 0.01

Values are presented as the mean±SD.
BMD: bone mineral density; GC: glucocorticoids;
a: patients without systemic GC therapy who received less than a 0.5g cumulative dose of prednisolone
b: patients with sytemic GC therapy who received more than a 10g cumulative dose of predonisolone

bone and induces development of pathologic fractures according to multiple regression analysis (Table 3, 5), and that patients with continuous systemic GC therapy have both low trabecular and cortical BMD and numerous fractures according to a comparison between groups (Table 6). The findings of this report confirm that GC administration causes both types of bone loss and pathologic fractures[8].

The present study has also shown that systemic GC administration decreases cortical bone volume and bone strength (Table 4, 6). Given that both cortical bone density and cortical bone volume provide bone strength[4, 5, 7, 11, 15], the GC-induced bone strength loss and fractures appear to correlate with the reduction in both cortical density and cortical volume.

Aging is reportedly associated with a reduction in cortical volume due to a widening of the marrow cavity as well as a reduction in cortical density[10-12, 16]. Using pQCT, Fujii et al. previously reported that the radius cortical volume and density are rectilinearly correlated in the aging processes of healthy adult humans[11]. Similar findings have also been reported in secondary hyperparathyoidism in uremic patients on maintenance hemodialysis[17]. To date, no studies have

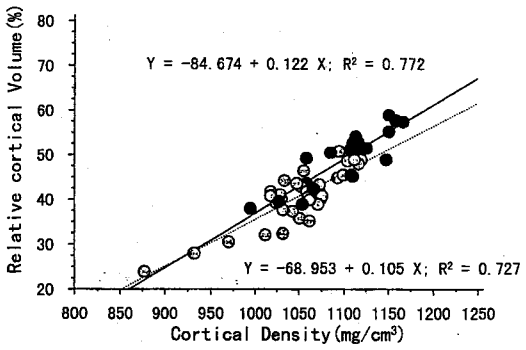

Fig. 1. Relationship between cortical density and relative cortical volume in patients with asthma.

A highly significant correlation ($p < 0.001$) of $r^2 = 0.727$ was obtained in 36 patients with systemic glucocorticoid therapy who received more than a 10g cumulative dose of predonisolone (◍). A highly significant correlation ($p < 0.001$) of $r^2 = 0.772$ was also obtained in 24 patients without systemic glucocor-ticoid therapy (●). Relative cortical volume (%) and cortical BMD showing a rectilinear relationship was expressed as $Y = 0.105(\pm 0.011)X - 68.953(\pm 11.881)$ in the 36 patients with systemic GC therapy, and $Y = 0.122(\pm 0.014)X - 84.674(\pm 15.447)$ in the 24 patients without systemic GC therapy.

examined whether this correlation is valid for patients who suffer from GC-induced bone loss.

We have demonstrated that the radius cortical bone volume and density are rectilinearly correlated in asthmatic patients, regardless of the level of systemic GC therapy (Figure 1). The two cortical volume-density slopes in Figure 1 of patients with or without continuous systemic GC therapy appear largely identical. These slopes are also roughly identical to the slope for healthy adult humans in the previous repor[11]. In view of the highly significant correlation between relative cortical volume and density in the radial cortex, a single process of GC-induced cortical bone resorption, or so-called cortical porosity, appears to be responsible at both the endosteal and intracortical sites for causing similar and simultaneous decreases in cortical bone volume and density, as in the aging processes[11], or in secondary hyperparathyoidism in uremic patients on maintenance hemodialysis[17].

Although it is still difficult to non-invasively image three-dimensional cortical bone micro-architecture, as in microcomputed tomography (μCT) imaging of trabecular bone[9], we believe that the progress made for various techniques will make such imaging possible in the future, and that imaging of cortical bone micro-architecture will contribute to the prediction of biomechanical properties.

In conclusion, systemic GC administration decreases both cortical bone density and cortical bone volume, and ultimately decreases bone strength. GC administration appears responsible for the process of cortical bone porosity at both the endosteal and intracortical sites, and thus causes similar and simultaneous decreases in cortical bone volume and density. This cortical bone porosity ultimately contributes to GC-induced bone strength loss and fractures.

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ステロイド依存性気管支喘息症例の病的骨折と皮質骨傷害の検討

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【目的】骨強度には皮質骨の状態が主に関与するため，全身ステロイド療法に伴う病的骨折と皮質骨障害について検討した。【方法】閉経後女性気管支喘息患者82例で，ステロイド依存性群と非依存群において，椎体骨折数，橈骨皮質骨骨密度，皮質骨容量，骨強度（SSI）をX線側面像とpQCTを用いて検討した。【結果】ステロイド依存群はステロイド非依存群に比べて，椎体圧迫骨折数は有意に多く，橈骨皮質骨骨密度，皮質骨容積比，骨強度は各々有意に低下していた。また，皮質骨骨密度と容積は一定の割合で減少していた。椎体圧迫骨折数は皮質骨骨密度，皮質骨容積比，骨強度の全てと高い相関を認めた。【結論】ステロイド依存性気管支喘息症例においては，皮質骨の骨密度と容量が同時に減少して骨強度が減少することによって，病的骨折が発症すると考えられた。