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Publication Date

2021-02-01

DOI

10.1016/j.bone.2020.115604

Peer reviewed

HHS Public Access

Author manuscript

Bone. Author manuscript; available in PMC 2022 February 01.

Published in final edited form as:

Bone. 2021 February ; 143: 115604. doi:10.1016/j.bone.2020.115604.

Regional Variation in Bone Turnover at the Iliac Crest versus the Greater Trochanter

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Abstract

Background: Iliac crest bone biopsy with histomorphometry is the gold standard for diagnosis of abnormalities in bone turnover, yet fractures more frequently occur at the greater trochanter of the hip. Whether bone turnover is similar at these two anatomic sites within individuals is uncertain.

Methods: We collected bone biopsy samples from the ipsilateral iliac crest and greater trochanter in 9 deceased individuals undergoing autopsies at an academic medical center between March-August 2018. We measured 14 static bone histomorphometry parameters including osteoclast number (N.Oc/T.A), eroded surface (ES/BS), trabecular separation (Tb.Sp), osteoclast surface (Oc.S/BS) and osteoid volume (OV/BV) as markers of bone turnover, mineralization, and volume (TMV), and evaluated the correlation of these markers between the iliac crest and greater trochanter.

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CRediT Author Statement

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Results: Average age at time of death was 58±15 years, 2 were women, and average time from death to autopsy was 2.9 ± 1.8 days. Overall, correlations of the markers of bone turnover across the two sites were poor, ranging from as low as 0 for Tb.Sp (p=1.0) to as high as 0.583 for Oc.S/BS (p=0.102).

Conclusions: Static histomorphometric measures of bone turnover at the iliac crest may not provide reliable information about turnover at other anatomic sites.

INTRODUCTION

Hip fractures are common, costly, and strongly associated with subsequent morbidity and mortality.[1–3] Postoperative mortality rates approach 30% during the first year following hip fracture.[4–6] In the general population, individuals with fracture history over the age of 50 have a >50% higher risk of hip fracture.[7] Specifically, among older post-menopausal women who participated in the Study of Osteoporotic Fractures (SOF), a prior fracture of any bone after age 50 was associated with a 68% higher risk (95% CI: 1.44–1.95) of hip fracture.[7]

In cases of repeat fractures, patients are referred for a clinical work-up to determine the most appropriate treatment plan to improve bone quality and reduce fracture risk. In addition to addressing non-skeletal factors, such as falls, that contribute to fracture occurrence, bone biopsies can be obtained to determine the underlying bone pathology and guide treatment plans. Bone biopsy and histomorphometry have traditionally been done at the iliac crest because this area is easily surgically accessible, and is non-weight bearing, and normative ranges in healthy individuals are available facilitating quantitative assessment of the results. Although bone biopsy at the iliac crest is considered the gold standard for determining bone turnover, mineralization, and volume (TMV) in patients with chronic kidney disease,[8] the ilium is rarely the site of fracture. The consistency of bone TMV at the iliac crest with other anatomic sites has been questioned in prior studies.[9–16] Specifically in human cadavers, Hiller and colleagues compared bone volume (BV/TV) between the iliac crest, proximal tibia, and lumbar spine.[9] And Podenphant et al. compared bone formation rate (BFR) between 24 skeletal sites within a single individual.[10] Both of these studies suggested that bone quality between sites is not always consistent.[9, 10] Thomsen, Amling, and colleagues have found similar results.[11, 15] Other studies, however, suggest higher correlations. For example, Mellish and colleagues found that the trabecular bone of the ilium has similar remodeling activity to the vertebral body, perhaps because these are both red marrow skeletal sites.[13, 16, 17] This finding did not extrapolate to other skeletal sites such as the greater trochanter, where little is known about its remodeling activity even though it is the site most commonly involved in hip fractures.

We hypothesized that taking bone tissue and performing histomorphometric assessment at a common site of fracture (the greater trochanter) may deliver more relevant information about bone metabolism and structure, and may guide appropriate secondary fracture prevention therapy. Because the hip is a common site of fracture, we sought to determine whether static measures of bone TMV at the greater trochanter were correlated with these same bone parameters at the iliac crest within autopsy cases in at our academic medical center.

METHODS

We collected bone tissue samples from the ipsilateral iliac crest and greater trochanter in 11 deceased individuals undergoing autopsies at the University of California, San Diego (UC San Diego) between March and August 2018. The UC San Diego Human Research Protections Program / Institutional Review Board approved the collection of tissue samples and relevant clinical data for this study.

Iliac crest biopsies were collected according to standard procedures where autopsy patients were positioned supine, and a 1cm incision was made 2cm posterior of the anterior superior iliac spine (ASIS). Using the Cheng large trephine and safety slide (Innomed, Inc., Atlanta, GA), the investigators directed the slide and trephine toward the umbilicus to collect a bicortical sample of the iliac crest. Greater trochanter biopsies were collected with the patient in the supine position and the lower extremity rotated to neutral. Following palpation of the greater trochanter, a 1–2cm incision was made 1cm distal to the greater trochanter, at the level of the lesser trochanter, where the Cheng safety slide and trephine were directed toward to the umbilicus to follow the long-axis of the femoral neck.

The Cheng trephine provided biopsies approximately 1cm long x 0.5cm in diameter. Upon collection, bone tissue was immediately placed in >70% ethanol and stored in a 4°C refrigerator and later shipped to the University of California Los Angeles (UCLA) Bone Histomorphometry Core Laboratory for processing and analysis. Specimens were batched and sent altogether, and specimens were labeled in a way that blinded the pathologist from knowing which specimens were pairs from the same individual. After fixation in 70% ethanol and dehydration in 100% ethanol followed by overnight incubation in xylene, the bone biopsy sample was infiltrated with plastic mixture (methyl methacrylate/dibutyl phthalate and benzoyl peroxide) for 9 days/9 solution changes. The sample was then embedded in 2.5% benzoyl peroxide overnight at 37°C. The undecalcified sample was trimmed and 1/3 of the core was cut off and the center core samples were sectioned using a Supercut 2065 microtome equipped with a tungsten carbide knife. Twelve slides were then prepared from serial sections, apart by 25 um. Two slides containing 5 um sections of undecalcified bone were stained with Toluidine blue (slide number 4 and slide number 8) for histological evaluation of trabecular and cortical bone structure. Both of the slides were carefully observed in the microscope and no gross difference was observed. Slide number 8 was used for analysis. All trabecular/cancellous bone was measured. Primary and derived bone histomorphometric parameters were assessed in trabecular bone under 200x magnification using the OsteoMetrics® system (OsteoMetrics). The bone marrow cell, osteoblast, and osteoclast were well identified. These methods have been published previously.[18, 19] Bone histomorphometric parameters were computed and described according to recommendations of the ASBMR Histomorphometric Nomenclature Committee.[17, 20, 21]

Specific bone histomorphometry parameters measured are listed in Tables 2 and 3. As patients were deceased and had no clinical indications for bone biopsy prior to death, none had received tetracycline bone labeling. Thus, we were unable to assess dynamic histomorphometry parameters, such as bone formation rate (BFR) or mineral apposition rate

(MAR).[17] We observed that the post mortem status resulted in osteoblast death if not obtained within 48 hours which precluded assessment of osteoblast number and osteoblast surface ratios as our samples were collected post mortem. As only one individual had osteoblasts, we did not include osteoblast measurements in our analysis. We were able to assess osteoclast number and osteoclast surface ratios, however, which is consistent with prior work showing osteoclasts are detectable in bone marrow for up to 7 days post mortem.[22]

We evaluated the correlation of the 14 bone histomorphometry parameters between the iliac crest and greater trochanter using Spearman correlations. As bone parameters are altered in chronic kidney disease (CKD),[23] we evaluated the subset of 3 patients with CKD separately. P-values < 0.05 were considered statistically significant. All analyses were performed using SAS 9.4 (SAS, Cary, NC).

RESULTS

Among 11 autopsy cases, we were unable to collect a bone biopsy from the greater trochanter in one, and unable to measure bone histomorphometry parameters in the greater trochanter specimen in another individual, leaving an analysis dataset of 9 individuals. These individuals had a mean age of 58 ± 15 years, 2 (22%) were female, and 7, (78%) were White. Two (22%) had diabetes, 3 (33%) had an eGFR < 60 mL/min/1.73m^2, and 7 (78%) had ever smoked. The average number of days between death and autopsy and bone biopsy was 2.9 ± 1.8 days with a post-mortem interval range from 1 to 6 days (Table 1).

Table 2 depicts Spearman correlation coefficients for 14 bone histomorphometry parameters at the iliac crest compared to the greater trochanter in the 9 individuals. Correlations ranged from as low as 0 for trabecular separation (Tb.Sp) (p=1.0) to as high as 0.583 for osteoclast surface (Oc.S/BS) (p=0.102); thus overall there was no, or at most minimal, correlation of these measures across the two anatomic sites. For side-by-side visual comparison of the greater trochanter and iliac crest biopsies within one individual, Figures 1a and 1b present 5 um Toluidine blue stain sections for each site; and Figures 1b and 1c present these samples magnified.

Table 3 presents the 14 bone histomorphometry parameters at the iliac crest and greater trochanter for the subset of 3 individuals with eGFR < 60 mL/min/1.73m^2. These individuals had mean eGFR 33.3 ± 24 mL/min/1.73m^2; one with Stage 3a, one with Stage 3b, and one with Stage 4 CKD. Average serum creatinine was 2.9 ± 2.4 (mg/dL), serum calcium was 8.4 ± 0.4 (mg/dL), and serum phosphorus 3.4 ± 1.1 (mg/dL). Serum parathyroid hormone (PTH) was not measured in these individuals. There were not enough cases to determine correlations, but descriptive analyses highlight the differences in the measured parameters between the iliac crest and greater trochanter in this subset as well. The discrepancy in osteoclast-related parameters between the iliac crest and greater trochanter appeared to persist even in this subset with CKD.

DISCUSSION

Comparing bone obtained at the iliac crest to that obtained from the greater trochanter in 9 autopsy cases, we found absent or very weak correlations between 14 static measures of bone turnover by histomorphometry.

Bone biopsy and histomorphometry at the iliac crest have been the gold standard in research and clinical care for evaluating measures of bone TMV in patients with CKD.[8] Yet our findings, along with those of others, illustrate that bone parameters at one anatomic site are not always representative of those at other anatomic sites.[9–11] Specifically, Podenphant and colleagues studied a single older woman who had received tetracycline labeling for bone biopsy, but died before the surgical procedure, thereby allowing extensive bone biopsy at multiple anatomic sites at autopsy, and evaluation of both static and dynamic markers of bone turnover. Comparing bone specimens at 24 anatomic sites including bilateral iliac crests, thoracic and lumbar vertebrae, and bilateral upper and lower extremities, among others, they found that the between site variance was significantly larger than the within site variance for apposition rate (M), fraction of labeled surface (Flab sur%), and bone formation rate (BFR).[10] Hiller and colleagues evaluated bone volume (BV/TV) from the iliac crest, lower lumbar vertebra, and proximal tibia in 12 embalmed cadavers, and similarly concluded that trabecular bone volume results of one site are not always representative of that of other sites.[9] Similarly, Thomsen and others investigated static bone histomorphometry parameters at the iliac crest and lumbar vertebra in 24 male and 24 female autopsy specimens, and found that age related changes occurred at both sites, but the correlations of static histomorphometric parameters, e.g. bone volume BV/TV, trabecular thickness (Tb.Th), and trabecular separation (Tb.Sp), between the iliac crest and vertebrae were only weakly correlated. Relationships between the iliac crest and greater trochanter are unexplored. Thus, we confirm these findings, and extend them in important ways. We showed that histomorphometric parameters at the iliac crest were not correlated with the same parameters at the greater trochanter. In order to better understand bone TMV at the greater trochanter, it may be valuable in future work to utilize bone from this site for histomorphometry, particularly if bone is accessible at the time of surgical repair of fracture.

Clinically, bone biopsies are most frequently obtained in patients with CKD, since high turnover and low turnover disease states are more common in CKD, above and beyond age-related osteoporosis. We describe the 14 static bone histomorphometry parameters in the 3 individuals with eGFR < 60 mL/min/1.73m^2. We were particularly interested in osteoclast parameters, as prior work in our lab suggest they correlate well (AUC > 0.78) with bone turnover by tetracycline labeling when evaluated at the iliac crest (unpublished data). While we were unable to test correlations between the iliac crest and greater trochanter in this small subset, we observed differences in osteoclast-related parameters between the iliac crest and greater trochanter within the subset with eGFR < 60 mL/min/1.73^2, suggesting that those with lower eGFR have lower levels of osteoclast-related parameters. This preliminary finding will require confirmation in larger studies in the future.

This study has several limitations. Given delays in obtaining family and next-of-kin consent for autopsies, we were unable to obtain biopsy within 24 hours post-mortem in most cases,

which prevented the measurement of osteoblasts. We observed that osteoblasts had frequently degraded within this time frame. Further, because these samples were collected as part of autopsies, we were unable to perform tetracycline labeling to determine whether dynamic bone turnover markers, e.g. bone formation rate, mineralization formation rate and mineralization lag time, also differed between the iliac crest and greater trochanter. So, we were unable to fully characterize bone turnover and mineralization via dynamic parameters. However, since osteoclast surface (Oc.S/BS) is correlated with bone turnover by dynamic measures, the low correlation of static markers of osteoclasts observed here suggest that the dynamic markers of bone turnover may differ by anatomic site as well. We recognize that there are currently no normative values for bone turnover at the greater trochanter, which prevents evaluation of whether individuals had high or low bone turnover at this site. Establishing normative ranges should be a high priority, which we are currently pursuing. Lastly, the study sample is small, limited to two anatomic sites, and recruited from a single academic medical center, and only 3 individuals had CKD.

CONCLUSIONS

We found poor agreement of static bone histomorphometry parameters between the iliac crest and greater trochanter in 9 human autopsy specimens. These data suggest that bone histomorphometric measures of the iliac crest may not always provide reliable information about bone turnover at the hip; the anatomic site where fractures frequently occur. Future studies are needed to determine whether bone biopsies from other anatomic sites, particularly those that are prone to fracture, provide more specific information about underlying bone pathology. These studies may ultimately guide approaches to treatment to prevent re-fracture.

ACKNOWLEDGEMENTS

The authors would like thank Koichi Masuda, MD, Pranav Garimella, MD, Craig Gibson, Diener, and the UC San Diego Pathology Faculty, Staff, and Residents for their assistance with collecting bone tissue in the autopsy suite at UC San Diego.

Funding for this project was provided by the National Heart, Lung, and Blood Institute (K01 HL122394 and R03 HL146875), the National Institute on Aging (R01 AG065876), and the National Institute of Diabetes and Digestive and Kidney Diseases (K24 DK110427, F32 DK116476) at the National Institutes of Health.

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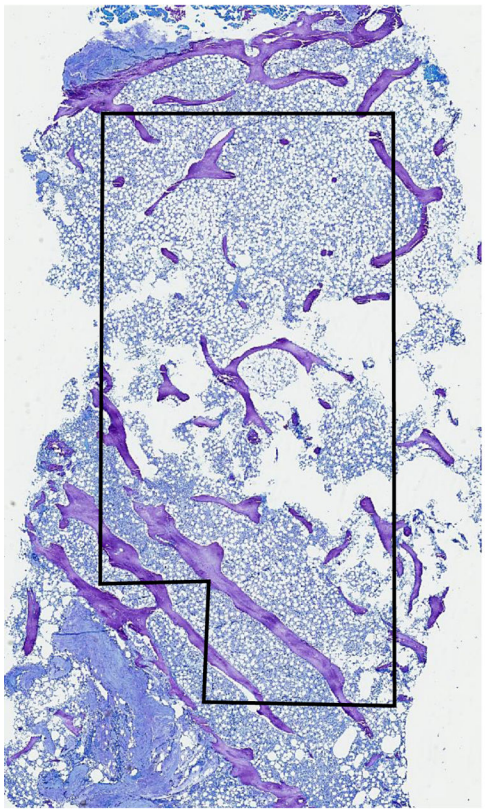
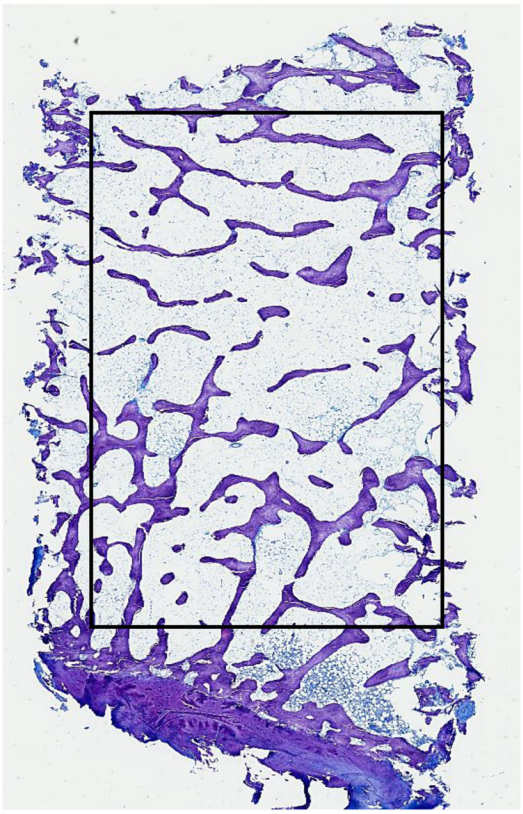
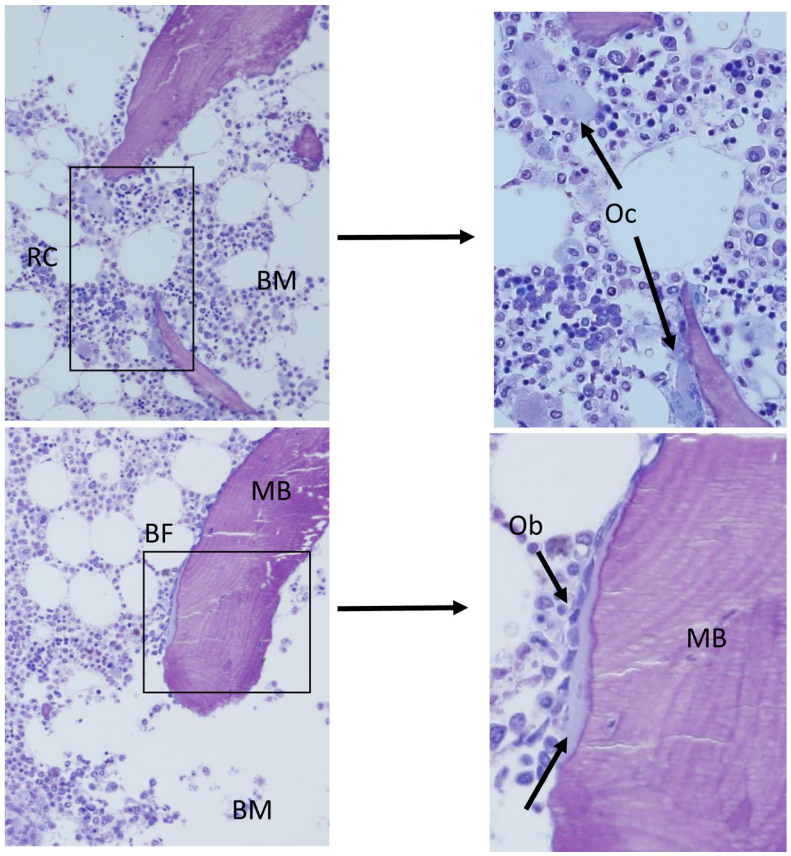
1a. Iliac Crest



1b. Greater Trochanter



1c.
Iliac Crest
Magnified



MB: Mineralized Bone
BF: Bone Formation Site
O: Osteoid
Ob: Osteoblasts
Oc: Osteoclasts

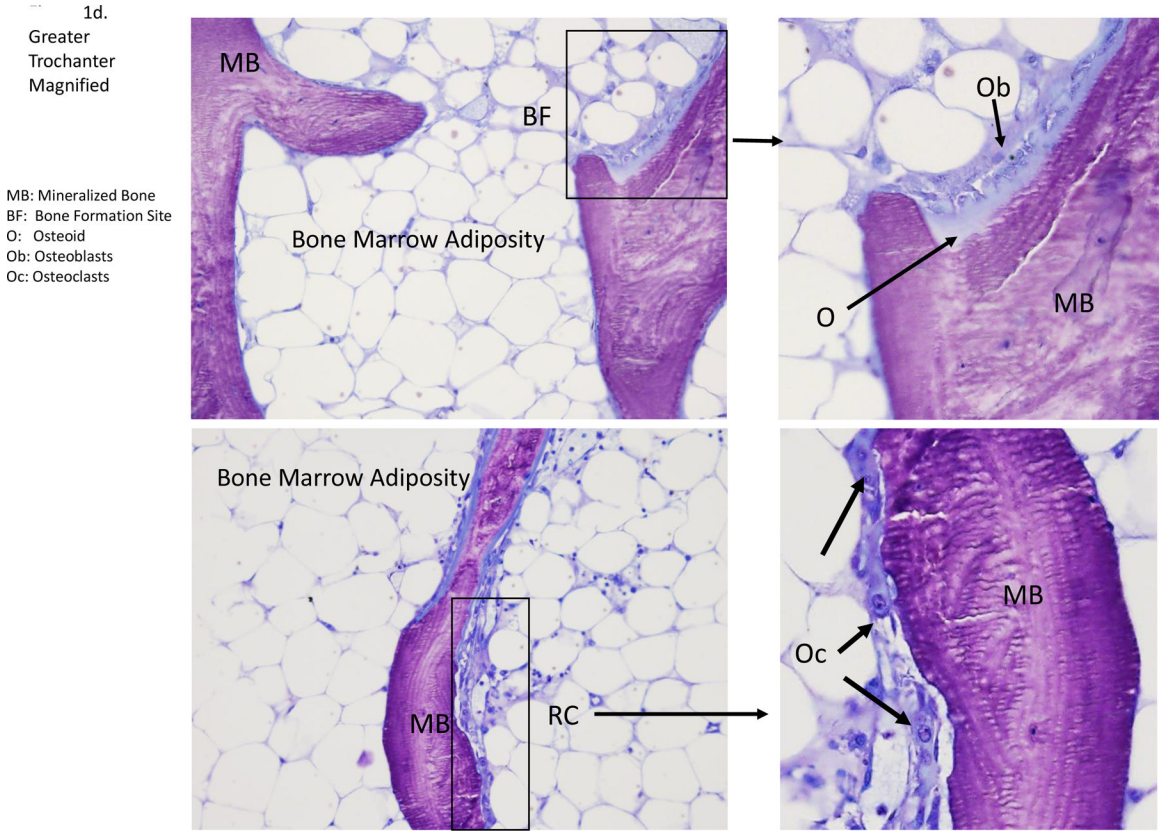


Figure 1.
Bone biopsy of the iliac crest and greater trochanter from a representative subject

Table 1.

Clinical characteristics of 9 autopsy cases with static bone histomorphometry parameters measured at the iliac crest and greater trochanter

POPULATION CHARACTERISTICS	n=9
Age, years, mean (SD)	58.8 (15.5)
Sex, n, % female	2, 22%
Number of days between death and biopsy, mean (SD)	2.9 (1.8)
Diabetes prevalence, n, %	2, 22%
eGFR <60 mL/min/1.73m^2, n, %	3, 33%
Ever smoker, n, %	7, 78%
White Race, n, %	7, 78%
Height, m, mean (SD)	1.8 (0.1)
Weight, kg, mean (SD)	87.9 (17.1)
BMI, kg/m^2, mean (SD)	28.5 (5.8)
Taking anti-resorptive medications, n, %	0, 0%

Table 2.

Static bone histomorphometry parameters at the iliac crest compared to the greater trochanter in everyone (n=9)

	Iliac Crest*	Greater Trochanter*	Spearman Correlation	p-value
T.Ar, mm^2	43.8 (15.7)	54.7 (24.9)	0.217	0.590
B.Ar, mm^2	6.5 (1.9)	8.6 (2.8)	−0.183	0.637
Cn-BV/TV, %	15.9 (5.3)	17.4 (6.1)	0.083	0.838
Tr.Th, mcm	108.7 (32.3)	118.7 (50.7)	0.167	0.680
Tb.Sp, mcm	600.3 (154.4)	562.9 (149.4)	0	1.0
Tb.N, /mm	1.5 (0.3)	1.5 (0.3)	−0.500	0.179
OV/BV, %	0.2 (0.1)	0.1 (0.1)	−0.326	0.426
OS/BS, %	2.5 (2.3)	1.0 (1.2)	−0.494	0.185
O.Th, mcm	3.1 (1.3)	2.4 (0.9)	−0.257	0.649
ES/BS, %	1.2 (1.2)	0.5 (0.7)	0.383	0.322
Oc.S/BS, %	0.8 (0.9)	0.3 (0.4)	0.583	0.102
N.Oc	14.0 (14.6)	10.4 (19.2)	0.328	0.405
Oc.N/T.A, $/mm^2$	0.3 (0.3)	0.1 (0.2)	0.339	0.387
N.Oc/B.Pm, /mm	0.1 (0.2)	0.1 (0.1)	0.170	0.675

*Mean (SD)

**T.Ar (mm^2) = tissue area, B.Ar (mm^2) = bone area, Cn-BV/TV (%) = cancellous bone volume, Tb.th (mcm) = trabecular thickness, Tb.Sp (mcm) = trabecular separation, Tb.N (/mm) = trabecular number, OV/BV (%) = osteoid volume, OS/BS (%) = osteoid surface, O.Th (mcm) = osteoid thickness, ES/BS (%) = eroded surface, Oc.S/BS (%) = osteoclast surface, N.Oc = osteoclast number (in 2D) Oc.N/T.A ($/mm^2$) = osteoclast number (in cancellous bone), N.Oc/B.Pm (/mm) = osteoclast number/bone perimeter

Table 3.

Static bone histomorphometry parameters at the iliac crest compared to the greater trochanter in cases with CKD (n=3)

	Iliac Crest*	Greater Trochanter*
T.Ar, mm^2	47.9 (20.4)	62.7 (21.5)
B.Ar, mm^2	7.0 (2.6)	10.2 (2.9)
Cn-BV/TV, %	15.8 (5.1)	16.6 (1.2)
Tr.Th, mcm	113.3 (15.6)	133.8 (43.0)
Tb.Sp, mcm	644.8 (204.5)	675.9 (229.4)
Tb.N, /mm	1.4 (0.3)	1.3 (0.4)
OV/BV, %	0.1 (0.1)	0.1 (0.1)
OS/BS, %	2.6 (2.0)	1.3 (1.7)
O.Th, mcm	3.2 (2.0)	2.2 (0.8)
ES/BS, %	0.4 (0.2)	0.1 (0.1)
Oc.S/BS, %	0.1 (0.1)	0.04 (0.04)
N.Oc	2.3 (2.1)	1.3 (1.5)
Oc.N/T.A, $/mm^2$	0.2 (0.3)	0.01 (0.02)
N.Oc/B.Pm, /mm	0.1 (0.1)	0.005 (0.009)

*Mean (SD)

**T.Ar (mm^2) = tissue area, B.Ar (mm^2) = bone area, Cn-BV/TV (%) = cancellous bone volume, Tb.th (mcm) = trabecular thickness, Tb.Sp (mcm) = trabecular separation, Tb.N (/mm) = trabecular number, OV/BV (%) = osteoid volume, OS/BS (%) = osteoid surface, O.Th (mcm) = osteoid thickness, ES/BS (%) = eroded surface, Oc.S/BS (%) = osteoclast surface, N.Oc = osteoclast number (in 2D) Oc.N/T.A ($/mm^2$) = osteoclast number (in cancellous bone), N.Oc/B.Pm (/mm) = osteoclast number/bone perimeter