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Bone loss after stroke over 52 weeks at os calcis: influence of sex, mobility and relation to bone density at other sites

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Abstract

Background: as life expectancy increases after stroke, skeletal consequences become increasingly important, and patients at risk of fracture require identification. We have investigated peripheral bone mineral density (BMD) measurement at the heel as a possible surrogate for hip dual energy X-ray absorptiometry measurement and we have related bone loss over 52 weeks to balance and mobility.
Methods: BMD at the heel (PIXI), proximal femur and whole body (QDR4500A), Tinetti (a measure of mobility and balance) and Barthel (a measure of activities of daily living) scores were measured in 52 patients (27 males and 25 females) within 8 weeks of stroke and repeated in 27 (15 males and 12 females) after 52 weeks.
Results: BMD was not initially low at the femoral neck (FN). A significant fall occurred on the stroke side (SS) over 52 weeks at the heel, FN and total hip (TH), in both sexes, but was greater in women. On the non-SS, women lost bone at the TH and heel. Patients who were in the lowest Tinetti score tertile initially, showed significant loss of bone in the FN (14.5%) and at the heel (12.2%) on the SS. BMD at the SS heel correlated with the FN at 8 weeks ($r = 0.64$, $P<0.01$) and at 52 weeks ($r = 0.60$, $P<0.01$) after stroke. Women lost more bone than men on SS but also lost bone on the non-SS at the same sites, suggesting that SS bone loss may result from factors additional to stroke.
Conclusion: heel BMD was a useful surrogate for hip BMD. Low initial Tinetti scores were an indicator of bone loss and, together with initial BMD measurements, provide a useful indication for those needing early prophylaxis against bone loss.

Keywords: *bone density, gender, heel, mobility, stroke, elderly*

Introduction

Stroke is a recognised risk factor for fracture and may account for 10% of all admissions for hip fracture [1]. With the trend towards increasing stroke occurrence [2, 3], and longer survival after stroke [2], this proportion may increase along with the population at risk. Women with stroke may have lower initial bone mineral density (BMD) [4] compared with a healthy population, and bone density may change most rapidly early in the post-stroke period [5]. Early identification of those who might benefit from treatment to prevent fracture is therefore important. BMD in the non-stroke population predicts the likelihood of future fracture prevention following bisphosphonate administration [6]. Because dual energy X-ray absorptiometry (DXA) of the hip may be difficult to perform early after stroke, peripheral scanning may be useful. Studies at the heel in stroke have largely used ultrasound [7], and bone density at the heel has variously been described as normal or low [8, 9]. Ankle oedema in stroke patients may be a confounding factor leading to these contradictory data [10] but one unlikely to have any effect with DXA. Although the reported changes at the heel are less than those at the proximal femur using DXA [7], heel DXA is less burdensome for a patient and could be undertaken by trained staff on a stroke unit. We have therefore compared BMD changes at the heel and hip using DXA to investigate the value of this technique in following bone loss after stroke.

Mobility following stroke may also play a role in determining bone loss on the stroke side (SS). In a cross sectional

study, stroke patient mobility correlated with stiffness of bone at the os calcis [8], and elsewhere, change in BMD at Wards triangle over 12 months correlated with motor function post-stroke [11]. We have therefore investigated the relationship between both a performance-oriented evaluation of balance and gait (Tinetti) and an index of functional ability (Barthel) and BMD changes at the proximal femur and at the heel.

Methodology

Subjects

Patients were assessed for their suitability in the stroke unit. Subjects were excluded if they had non-vascular causes such as tumour or if they had a transient ischaemic attack. Assessment was at baseline (up to 4 weeks post-stroke; mean time to assessment 1.62 weeks, range 0.57–3.86 weeks) and at 8 weeks (stage 1) and 52 weeks (stage 2) post-stroke. Of 52 acute stroke patients (27 males and 25 females) originally enrolled, 25 patients were unavailable to proceed beyond stage 1. Of these, 10 were unwilling or unable to continue, and 15 were unable to be contacted. This left 27 patients (12 females and 15 males), followed up to stage 2.

All subjects who took part in the study were 60 years and over (mean age 73.3 years; males, 72.9 years; females, 73.8 years) and were given written information about the study. Local research ethics committee approved the study information. Informed consent was obtained at baseline from the patient (in over 90% of cases), or otherwise agreement was gained from a relative or carer. Further consent for follow-up was obtained from patients at stage 1.

Data collected at the time of the assessment included date and site of past fracture, drug history and date and side of stroke (hemiplegic or SS).

BMD

BMD at both heels was measured using a PIXI densitometer (GE Lunar, GE Healthcare) at baseline, stage 1 and stage 2. PIXI densitometer is a mobile scanning device that is transportable to the ward setting in the acute phase of stroke. At stages 1 and 2, BMD at both proximal femora, and bone mineral content (BMC) and BMD of the whole body (WB) were measured using a QDR4500A bone densitometer (Hologic Inc., Bedford, MA, USA). The T score (number of SDs the patient's BMD is from the peak bone mass) and Z score (number of SDs the patient's BMD is from age-matched mean) were derived from manufacturer's normal ranges. Proximal femur measures femoral neck (FN), trochanter, intertrochanteric, Wards triangle and total hip (TH). WB BMC was divided into both upper and lower limbs (LLs). In eight subjects with hip or knee prostheses, involved regions were excluded, but heel results were included.

Mobility

The Tinetti balance scale (TBS) was used, at baseline and stage 2, to assess characteristics associated with mobility and severity of the stroke. TBS [12] was applied using the balance section (most patients were unable to do the gait assessment). There were 10 questions scored to give 16 points maximum. The Barthel index [13] was used to determine the level of independence at baseline and stage 2.

Statistics

SPSS Version 12 (SPSS Inc., Chicago, IL, USA) was used for all statistical tests. Student's *t*-test was used for paired [SS and non-stroke-side (NSS) and longitudinal BMD changes] and unpaired (age and BMD in follow-up and non-follow-up groups). ANOVA was used to compare BMD values between each Tinetti or Barthel tertile. The non-parametric Wilcoxon rank test was used for comparing longitudinal changes in Tinetti and Barthel scores. Correlations between BMD and Tinetti or Barthel results were performed with Pearson and Spearman's rank test. To test for significance in the longitudinal percentage changes of BMD at the heel and proximal femur, least significant change (LSC $= 2 * \sqrt{2} * CV$) was calculated from published co-efficient of variation (CV%), for the heel (2.5%) [14], and for the proximal femur (TH 3.7%, FN 9.3%) [15]. No data are published for Wards triangle on the QDR4500A. For the QDR1000 (an earlier version of the densitometer), the LSC at Wards triangle is 12.11% compared with 5.9% at the FN [16]. Power estimation was made of the number of subjects needed to show a BMD difference at 52 weeks, between SS and NSS, of 10%, as shown by Jorgensen *et al.* [17]. Using the LSC measure of standard deviation at the FN, a minimum sample size of 20 (with a power of 90%), measured on the two occasions, was calculated to be required to show a significant change at the 5% level.

Results

Fifty-two patients were assessed at baseline and stage 1; of these, 27 patients consented to follow-up to stage 2. At stage 1, SS BMD at the heel correlated with FN BMD in men ($Heel_m = 0.62$ (FN BMD) + 0.1; $r = 0.63$, $P<0.01$) and in women ($Heel_f = 0.63$ (FN BMD) + 0.04; $r = 0.73$, $P<0.01$). Patients not consenting to return at stage 2 showed no differences in their baseline Tinetti or Barthel, or BMD measurements at stage 1, from those followed up (Table 1). Of those continuing, 11 males and seven females had altered muscle tone in the LL, and of the remaining nine subjects (four males and five females), seven were affected in the upper limb, and two had dysarthria and balance impairment. In 17 patients (seven males and 10 females), the SS was on the patient's dominant side.

SS heel BMD was significantly lower than the NSS at baseline, but heel BMD on each side did not change significantly between baseline and stage 1 in either sex. FN Z score was not low in either sex at stage 1. Five women and one man had FN T score less than −2.5.

At stage 2 (52 weeks), heel BMD correlated with FN BMD on the SS ($r = 0.60$, $P<0.01$). Men had lost bone at the heel and all sites in the upper femur, except Wards triangle on the SS (Table 2). In women, significant SS loss was found only at the heel, TH and FN (Table 2). In the same

Table 1. Baseline data for patients followed up for 52 weeks and those not consenting for follow-up or unable to be contacted

	Male (mean ± SD)			Female (mean ± SD)		
	Patients followed up (n = 14)	Patients not followed (n = 12)	P value	Patients followed up (n = 13)	Patients not followed (n = 13)	P value
Age (y)	72.4 ± 7.8	72.2 ± 6.6	NS	73.9 ± 7.2	74.1 ± 6.9	NS
SS Heel BMD (g/cm^2)	0.58 ± 0.16	0.63 ± 0.12	NS	0.49 ± 0.10	0.41 ± 0.11	NS
SS femoral neck BMD (g/cm^2)	0.80 ± 0.17	0.80 ± 0.11	NS	0.71 ± 0.10	0.63 ± 0.15	NS
Height (cm)	170.2 ± 8.6	169.0 ± 6.9	NS	162.8 ± 4.1	159.4 ± 3.2	0.04
Weight (kg)	72.9 ± 15.1	79.3 ± 14.0	NS	75.8 ± 11.2	66.1 ± 10.6	0.04
Mobility						
Tinetti score (median)	9	11	NS	2.5	1	NS
Barthel score (median)	13	15	NS	11	8	NS

Tinetti score was out of a maximum of 16, and Barthel score was out of a maximum of 20.
BMD, bone mineral density; NS, not significant; SS, stroke side.

Table 2. Longitudinal changes in bone mineral density (BMD) (mean percentage change) and mobility (median score) for stroke patients followed up over 52 weeks

BMD site	Male	Female	Male and female
Mean percentage change from stages 1 and 2			
SS heel	−7.0[a]	−8.1[a]	−7.4[a]
NSS heel	−1.7	−6.0[a]	−3.6[a]
SS total hip	−3.1[a]	−3.6[a]	−3.3[a]
NSS total hip	−1	−3.5[a]	−2.2[a]
SS femoral neck	−7.5[a]	−8.5[a]	−7.9[a]
NSS femoral neck	−3.8	−1.5	−2.7[a]
SS trochanter	−6.6[a]	−3.2	−4.3[a]
NSS trochanter	1.3	−1.5	−1.4[a]
SS intertrochanter	−4.2[a]	−3.1	−3.1[a]
NSS intertrochanter	1.1	−4.9[a]	−2.1[a]
SS upper limb BMC	−13.3[a]	−4.9	−10.4[a]
NSS upper limb BMC	1.4	−2.7	−0.02
SS lower limb BMC	−6.7[a]	−4.9[a]	−6.1[a]
NSS lower limb BMC	−4.2[a]	−3.4[a]	−3.9[a]
Whole body BMC	−2.9[a]	−2.8	−2.8[a]
Mobility–median scores			
Tinetti baseline	8.5	2.5	7
Stage 2	9	9	9
Barthel baseline	13	11	12
Stage 2	19	18	19

[a]Denotes a significant change over 52 weeks (P<0.05).
BMC, bone mineral content; NSS, Non-stroke side; SS, stroke side.

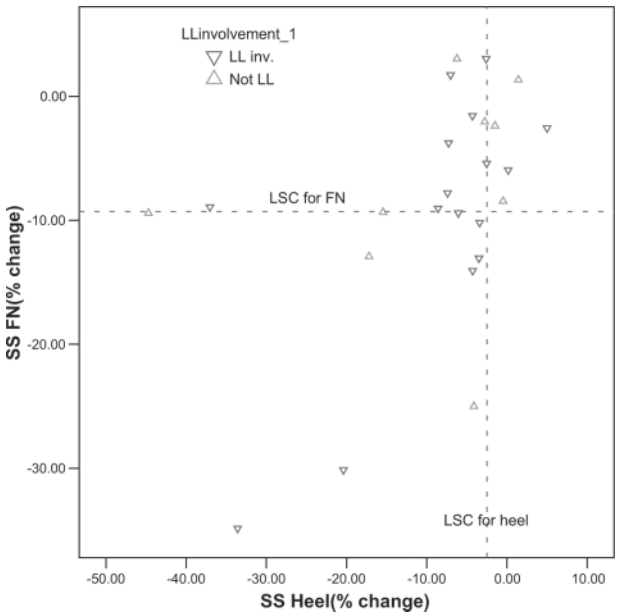

Figure 1. Percentage changes in heel bone mineral density (BMD) versus hip BMD between stages 1 and 2, showing least significant change (LSC) for heel (2.5) and hip (FN = 9.3). Patients with lower limb (LL) involvement appear as an inverted triangle ∇ and those without as an upright triangle Δ.

locations on the NSS, women showed a significant reduction at all sites except the FN and trochanter; men showed no loss.

Loss of BMD on the SS exceeded the LSC in 67 (male) and 83% (female) at the heel, 38 and 42% in FN and 46 and 50% at TH. All patients whose decline of FN BMD, over 52 weeks, was more than the LSC at the FN also exhibited a fall of heel BMD greater than the LSC at the heel. However, in those patients whose change of heel BMD exceeded the heel LSC, only 50% recorded a change at the FN which exceeded the LSC at that site (Figure 1).

WB BMC loss on the SS between stages 1 and 2, although greater in men compared with women, in both the upper limb and LL, did not differ significantly between the sexes (Table 2). In men, this loss, compared with NSS, was greater in the upper limb than in the LL (P<0.05).

SS BMD in eight patients taking drugs known to affect bone density (Bendrofluazide, Co-amilofruse, Frusemide and Risedronate) was not significantly different, at any stage or at any site, from those subjects not taking these drugs. BMD (stages 1 and 2) in subjects with muscle tone altered in the LL was the same as that in those without LL involvement at all sites including FN. No difference was found in BMD between SS-dominant and SS-non-dominant patients.

Tinetti score at the baseline correlated with the score at stage 2 (ρ = 0.47; P<0.05; Spearman rank), and median score

increased from 7 to 9 over 52 weeks in the sexes combined ($P<0.01$; Wilcoxon Rank). Patients were grouped into tertiles of Tinetti score at baseline [0–1 (female/male, 4/4); 2–9 (3/5); 10–16 (3/6)]. Those in the lowest tertile had a greater loss of BMD at stage 2, compared with the highest tertile, at the SS FN (Figure 2a) (sexes combined, $P<0.01$; males, $P<0.05$) and at the SS heel (Figure 2b) (males only, $P<0.05$). Loss of bone at the FN (sexes combined) was 14.5% in the lowest Tinetti tertile, compared with 3.1% in the highest tertile; corresponding losses at the heel were 12.2 and 8.4% (Figure 2a and b). Results of the Barthel assessment showed similar findings.

When comparing Tinetti score of those subjects with altered tone on their SS (at baseline) and those without, patients with LL involvement had a significantly lower Tinetti score ($P<0.05$) at baseline and stage 2.

Discussion

This study did not find that patients with stroke had low BMD at the outset [4]. The value of BMD lies in the recognition of patients, particularly those with low BMD, who would benefit from prophylaxis to prevent bone loss after stroke, because delay may allow significant loss of bone [5]. Bone, once lost, is difficult to replace, and this loss will contribute to hip fracture, maximal within 3–4 years after stroke [1, 18]. Heel scanning may be a useful surrogate for spine and hip scans at the outset. In women, heel density at the outset was found to correlate with density at the FN. From this relationship, the BMD value of 0.6 g/cm^2 at the FN (T score of −2.5) corresponds to a heel BMD of 0.42 g/cm^2, a value which approximates that of 0.39 g/cm^2 derived in a larger study of healthy women [19]. Women were more likely than men to have initial BMD less dense than T-2.5, a BMD level below which alendronate treatment is associated with a significant reduction in fracture occurrence [6].

Measurement of BMD change identifies patients who lose bone rapidly. To be significant, changes must exceed the LSC of the technique. Wards triangle is the most unreliable [16], whereas, the heel has the lowest LSC, with the FN in an intermediate position (see methods). Significant reductions were most frequent at the heel. Initial and stage 2 bone density at the heel correlated with BMD at the FN and TH. Change in BMD was, however, less well-associated, possibly because following immobilisation, different sites lose bone at different rates, often associated with individual circumstances including nutrition and mobility [7].

Bone loss following stroke has not been studied much [11, 17]. On the paretic side (whole LL), it has been suggested that this loss, of about 3.7%, ceases after 4 months, in a study which only lasted 6 months [5]. Bone deficit in the upper femur (from a WB scan) was reported as 12.7% in a Swedish study [11], but the present study indicates that loss of bone varies according to site (Table 2). In this study, the 6.7% lost over 52 weeks in the whole LL suggests that bone loss may continue beyond 4 months. Analysis of individual bones in the LL would give greater insight into relative loss of bone at cortical and trabecular sites.

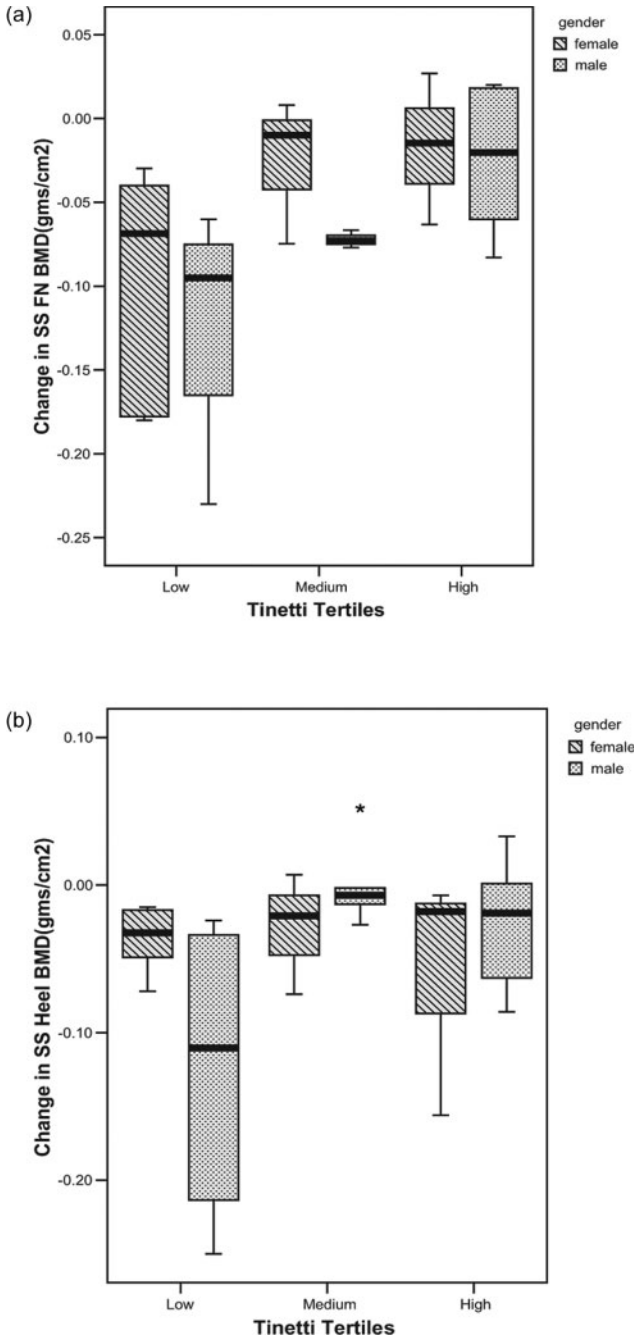

Figure 2. (a) Change in bone mineral density (BMD) (g/cm^2) at the femoral neck (FN), over 52 weeks, on the stroke side (SS), split into Tinetti tertiles: low (0–1), medium (2–9) and high (10–16). Change represents stage 2 minus stage 1, i.e. negative values are equivalent to a loss of BMD. (b) Change in BMD (g/cm^2) at the heel, over 52 weeks, on the SS, split into Tinetti tertiles: low (0–1), medium (2–9) and high (10–16). Change represents stage 2 minus stage 1, i.e. negative values are equivalent to a loss of BMD. *Indicates outlying datum point.

In contrast with an earlier study, we did not find an increase in NSS BMD [11]. Previous studies have not distinguished

between sexes [11]. Both sexes lose bone rapidly on the SS, but at most sites, this is generally greater in women (Table 2). However, much of this excess may result from non-stroke causes, because BMD generally declines more in females than in males, not only on the SS but also on the NSS. If the loss on the NSS is subtracted from the loss on the SS at stage 2, men appear to lose more than women at many sites. Women may suffer bone loss not only from immobilisation but also from ageing and sex hormone deficiency.

Bone loss after stroke is also influenced by mobility. Previous studies showed that confinement to a wheelchair in the first 2 months after stroke leads to a significant bone loss [17] and that the loss of function on the SS is associated with loss of bone density at Wards triangle [11]. The present data show that patients with the worst balance scores at the outset have a significant loss of bone not only at the upper femur but also at the heel over 52 weeks and only on the SS. Low Tinetti scores reflect poor mobility and an increased risk of falls and fall-related injuries [20]. Measurement, therefore, of BMD and balance at the outset will give useful information about the need for early prophylaxis against bone loss. Moreover, the heel, and possibly other peripheral sites such as the hand [21], may be suitable for monitoring, particularly in patients where access to axial measurement devices may be difficult in the acute stages of stroke or where BMD losses are expected to be larger than in the general population. This study is limited by small numbers and the possibility that subjects recruited may be those more likely to survive a stroke, although several showed very low mobility scores at the outset. Nevertheless, significant changes have been shown in BMD losses over 52 weeks.

Bone is lost in stroke patients by stage 2 and can be detected not only at the proximal femur but also at the heel. Use of this site for BMD measurements together with an assessment of balance or mobility at the outset may provide a means of identifying those patients at the greatest risk of having low bone density in the future. Prophylaxis could be particularly important in women in whom enhanced bone loss may result from factors other than stroke.

Key points

- Heel BMD may be a useful surrogate for the hip, in monitoring BMD changes in stroke, when combined with assessment of mobility and balance.
- Low mobility scores, measured soon after stroke, are an indicator of greater bone loss over time.
- A significant fall in BMD occurs over 52 weeks at the heel and proximal femur on the SS.

Conflicts of interest declaration

There are no conflicts of interest.

Declaration of sources of funding

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A cross-sectional survey of health state impairment and treatment patterns in patients with postherpetic neuralgia

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Abstract

Background: postherpetic neuralgia (PHN) develops in 8–24% of patients with herpes zoster. Few studies have evaluated the patient burden and treatment of PHN in general practice.
Objectives: to determine the patient burden of PHN with respect to pain intensity and impact on patient functioning and to characterise treatment patterns and health resource utilisation in general practice.
Methods: eighty-four patients with PHN were identified in general practice settings during an observational survey of neuropathic pain syndromes in six European countries. Patients answered a questionnaire that included: pain severity and interference items from the modified short form brief pain inventory (mBPI-SF); EuroQol (EQ-5D) survey; and questions related to current treatment, health status and resource utilisation. Physicians provided information on medications prescribed for PHN and pain-related co-morbidities (anxiety, depression and sleep disturbance).
Results: mean patient age was 71.0 ± 12.8 years, 76% were ≥65 years and 45% of patients had PHN ≥1 year. The mean pain severity index was 4.2, reflecting moderate pain despite 89% of patients taking prescription medications for PHN. Few medications with demonstrated efficacy against PHN (e.g. carbamazepine and gabapentin) were prescribed, often at suboptimal doses. Pain severity was associated with reduced EQ-5D health state valuation ($P<0.001$), greater pain interference on all domains ($P<0.001$) and increased health resource utilisation ($P = 0.008$).
Conclusions: PHN causes substantial patient burden expressed as interference with daily functioning and reduced health status associated with pain severity. This burden may result in part from suboptimal management strategies and suggests a need for more effective pain management.

Keywords: *postherpetic neuralgia, herpes zoster, neuropathic pain, patient burden, quality of life, health status, elderly*

Introduction

Herpes zoster (HZ) is characterised by painful blisters that erupt along a nerve path after reactivation of latent varicella zoster virus. HZ has an estimated incidence in the United States and Europe of 3.9–11.8/1,000 person-years in persons aged ≥60 years [1]. The acute pain of HZ significantly impacts patient function and quality of life [2, 3].

Postherpetic neuralgia (PHN), the most common complication of HZ, is a neuropathic pain frequently reported as lancinating, burning, shooting, stabbing, paroxysmal or electrical. It is often associated with abnormal sensory