



Skeletal consequences in patients after stroke

Zaburzenia szkieletowe u pacjentów po udarze mózgu

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Abstract

Loss of bone mineral density and fractures in patients after stroke are among the most serious health complications. Regarding serious post-stroke impediments, significantly contributing to elevated fracture incidence are falls. An increasing number of subjects after stroke have been observed and better understanding of the nature of skeletal changes might improve the efficacy of their management. The most important issues in post-stroke patients include an evaluation of bone loss magnitude, the occurrence of falls, fracture incidence and prevention procedures. An early start of pharmacological treatment plus exercise are recommended procedures in the early post-stroke stages, in order to diminish as much as possible subsequent bone loss, the direct consequences of which may include hemiosteoporosis, prolonged immobilisation, further falls and fractures. (*Pol J Endocrinol* 2011; 62 (1): 48–50)

Key words: bone densitometry, falls, fracture, stroke, therapy

Streszczenie

Niska gęstość mineralna kości oraz złamania u pacjentów po udarze to poważne powikłania. Bardzo istotny czynnik zwiększający występowanie złamań stanowią upadki. Liczba pacjentów po udarach wzrasta i zrozumienie natury zmian szkieletowych u chorych po udarach może w znaczący sposób wpłynąć na efektywność procesu terapeutycznego. Najistotniejsze kwestie to ocena wielkości obniżenia się gęstości mineralnej kości, zagrożenia upadkami, częstości występowania złamań i prowadzenie działań prewencyjnych. Podstawowe znaczenie ma wczesne wprowadzanie terapii farmakologicznej i ćwiczeń fizycznych w celu uniknięcia utraty tkanki kostnej skutkującej hemi-osteoporozą, długotrwałego unieruchomienia, upadków oraz złamań. (*Endokrynol Pol* 2011; 62 (1): 48–50)

Słowa kluczowe: densytometria, upadki, złamania, udar, terapia

Introduction

Loss of bone mineral density and fractures in patients after stroke are among the most serious health complications. Stroke is a medical condition which causes long-term disability and leaves 90 per cent of post-stroke subjects with certain functional limitations [1], including muscle weakness, pain, spasticity, cognitive dysfunction, poor balance and frequent falls. Reduced physical activity and immobilisation significantly influences the skeletal status [2]. The pathophysiology of post-stroke osteoporosis differs from other forms of the disease (post-menopausal, senile or secondary) and is more evident within the paretic side, involving the upper extremities more than the lower ones [3]. In general, hemiosteoporosis is observed, as bone loss occurs in one half of the skeleton. Among several causes with potential influence on the skeletal status, hemiplegia dura-

tion, the degree of functional recovery, vitamin D status, the use of anticonvulsants, the time period from stroke to stand-up, and pre-existing osteoporosis are bone loss triggering factors [4, 5].

A very serious complication of stroke is an increased risk of falls. Frequent falls may lead to high incidence of fractures, with the obvious fear of subsequent falls resulting in further reduction of what was already low physical activity, as well as in diminished social contacts, with interest in such contacts gradually being replaced by growing depression [1]. Both main consequences of stroke, bone loss and increased risk of falls, lead to an enhanced risk of fractures. Progressive hemiosteoporosis is a significant clinical finding because reports suggest that 4–15% of patients with hip fractures present with a history of previous cerebrovascular disorders and that 79–100% of the fractures occurred on the hemiplegic side [6, 7].



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With an increasing number of post-stroke subjects being recorded, a better understanding of the nature of skeletal changes may improve the efficacy of the patients' management. Skeletal changes in post-stroke patients have been evaluated in several studies [5, 8–17], the most important issues concerning bone loss magnitude, the occurrence of falls, fracture incidence and prevention procedures.

Bone loss after stroke

Bone loss is not uniform within the whole skeleton, with different degrees on the paretic side and with clear differences between upper and lower extremities, being usually more pronounced in the upper extremities [3]. Bone mineral density (BMD) may be compared in age and sex-matched controls or on the paretic vs. non-paretic side. Bone loss is commonly present on the paretic side; in the paretic arm, during the first year after stroke, it is the equivalent to at least two decades of bone loss in healthy individuals, and BMD of the non-paretic arm may also increase. Bone loss in the paretic lower limb can decrease by 10% in the first year, while being usually smaller in the other, non-paretic lower limb. In some evaluations, bone loss has been observed in cross-sectional and longitudinal studies. In a cross-sectional study, [12] decreases of 17% at the humerus and 12% at the femoral neck were observed and Jorgenson noted a 17% decrease at the paretic humerus with no change on the opposite side [10].

In longitudinal studies, loss in the paretic arm was 3.7% after one month, 12% after three months, and 17.4% after one year [18, 19]. In the non-paretic arm, BMD increased by 5.8% a year after a stroke [12].

Bone mineral density of the paretic lower limb decreased by 1% after a month, by 5% after three months [18] and by 12.2% a year after a stroke [12], while in the non-paretic lower limb, bone loss was 4% after a year [12]. No bone loss evaluations have been performed over post-stroke periods longer than one year.

Fractures after stroke

Fractures are common in stroke patients and occur mainly on the paretic side as the result of a fall. The significance of the problem was emphasised in a retrospective analysis of 568 patients with femoral neck fractures, of whom 27.4% had had a stroke [12]. In a longitudinal observation of 1,139 post-stroke patients, the risk of hip fracture increased 3.8 times in patients aged under 70 and 2.1 times in those above 79 [20].

Other authors have reported that fractures are more frequent in females, in patients below 70 and in cases of hemorrhagic rather than ischaemic stroke [21]. Data

from 273,288 hospitalised stroke patients indicated a seven-fold increased fracture risk during the first year after hospitalisation (mostly hip fractures) [22].

The risk of falls

Due to several factors mentioned earlier, the risk of falling is in post-stroke patients significantly increased. 73% of individuals who have had a stroke experience a falling incident within six months of their hospital discharge. In the six months following a stroke, an average of 3.4 falls are experienced [23]. Effective prevention of falls is essential for patients' management. The risk of falls rises with advanced age, male gender, in the presence of concomitant infections, in subjects who have a second stroke, post-stroke seizures, urinary incontinence, poor balance, various motor and mental disturbances, depression and visual problems secondary to stroke. Also the severity of stroke, the right hemispheric stroke, widespread white matter lesions and the use of drugs (analgesics, sedatives, hypertensives) may increase the frequency of falls.

Prevention strategies

An effective prevention strategy must take two main directions: firstly prevention of bone loss, and secondly prevention of falls.

An optimal prevention programme should include exercises, early stand-up, active rehabilitation and pharmacological treatment. Environmental modifications (removal of hazards, installation of grip bars, enhanced lighting) are needed; also important are mobility-related aids, changes of medications and the use of hip protectors.

Physical activity plays a very important role in post-stroke patients. Physical exercise can improve BMD and balance and reduce the risk of falls. Therefore, exercises are an essential element of the management of stroke-affected patients. Although the role of exercise is fairly obvious, no trial has yet been conducted with sufficient statistical power to show an exercise-associated reduction in fractures in post-stroke subjects. It has been estimated that such a trial, assuming fracture as the clinical end point for exercise intervention, would require 7,000 participants observed for at least five years, something that would be difficult to achieve in practice. Physical exercise improves muscle strength, preserving muscle mass. Forces acting on the skeleton during weight-bearing exercises may even exceed the levels observed during normal daily activity. The results of individual exercise groups indicate a growth of physical fitness, which may positively influence the status of bones. The participants improved their cardiovascular capacity, as

measured by oxygen consumption. The gain in bone health could be partially attributable to improved cardiovascular function. Only one, randomised, controlled trial has examined the effects of exercise on bone health in subjects after stroke. In a 19-week exercise programme, 32 patients received a variety of weight-bearing activities (sit-to-stand, stepping onto risers, brisk walking), leading to improved muscle strength, balance and aerobic fitness. The controls ($n = 3$) participated in a routine exercise programme. Femoral neck BMD did not change (-0.7%) on the paretic side and decreased in the controls by 2.5% [24].

No ideal exercise protocol has been devised which would effectively enhance bone health in patients after stroke with different disability levels.

Effective management (including physical exercise) ought to improve: balance, weight-bearing ability, gait, mobility, movement speed and postural control. Cheng et al. [25] verified the efficacy of exercises in a group of post-stroke patients. A group of 54 patients, 2-4 months after a stroke, received intensive physical training (30 minutes of symmetrical standing and 20 minutes of repetitive sit-to-stand exercises) five times weekly for three weeks.

During a six-month follow-up, only 16.7% of the patients experienced a fall, while in controls on conventional therapy, 41.7% recorded fall incidents [25].

Pharmacology in post-stroke patients may include vitamin D, active metabolites of vitamin D, bisphosphonates and calcium [4]. Risedronate is the only drug with proven reduction of fracture risk in subjects after stroke [26] and zoledronate can reduce bone loss [27].

Summary

The current guidelines for osteoporosis diagnostics and treatment do not take into account post-stroke osteoporosis. Post-stroke osteoporosis management should start with a careful and complex risk assessment. Residual walking deficit should be considered a key factor in osteoporosis risk assessment for the lower limbs. Pharmacological treatment plus exercise is the recommended procedure in the early stages after a stroke in order to minimise subsequent bone loss, which bears further risks of hemioosteoporosis, prolonged immobilisation and falls.

References

- Gresham GE, Fitzpatrick TE, Wolf PA et al. Residual disability in survivors of stroke-The Framingham Study. *NEJM* 1975; 293: 954-956.
- Donaldson ChL, Hulley SB, Vogel JM et al. Effects of prolonged bed rest on bone mineral. *Metabolism* 1970; 19: 1071-1084.
- Eng JJ, Pang MYC, Ashe MC. Balance, falls, and bone health: role of exercising in reducing fracture risk after stroke. *JRRD* 2008; 45: 297-314.
- Carda S, Cisari C, Invernizzi M et al. Osteoporosis after stroke: a review of the causes and potential treatments. *Cerebrovasc Dis* 2009; 28: 191-200.
- Beaupre GS, Lew HL. Bone-density changes after stroke. *Am J Phys Med Rehabil* 2006; 85: 464-472.
- Mulley G, Espley AJ. Hip fracture after hemiplegia. *Postgrad Med J* 1979; 55: 264-265.
- Poplingher AR, Pillar T. Hip fracture in stroke patients. *Acta Orthop Scand* 1985; 56: 226-227.
- del Puente A, Pappone N, Mandes MG et al. Determinants of BMD in immobilization: a study on hemiplegia. *Osteoporos Int* 1996; 6: 50-54.
- Iversen E, Hassager C, Christiansen C. The effect of hemiplegia on bone mass and soft tissue body composition *Acta Neurol Scand* 1989; 79: 155-159.
- Jorgensen L, Jacobsen BK, Wilsgaard T et al. Walking after Stroke: Does It Matter? Changes in Bone Mineral Density Within the First 12 Months after Stroke. A longitudinal Study. *Osteoporos Int* 2000; 11: 381-387.
- Jorgensen L, Jacobsen BK. Changes in Muscle Mass, Fat Mass, and Bone Mineral Content in the legs after stroke a 1 year prospective study. *Bone* 2001; 28: 655-659.
- Rammemark A, Nyberg L, Lorentzon R et al. Hemioosteoporosis After Severe Stroke, Independent of Changes in Body Composition and Weight. *Stroke* 1999; 30: 755-760.
- Sato Y, Maruoka H, Honda Y et al. Development of osteopenia in the Hemiplegic Finger in Patients with Stroke. *Eur Neurol* 1996; 36: 278-283.
- Sato Y, Maruoka H, Oizumi K. Amelioration of hemiplegia associated osteopenia more than 4 years after stroke by 1,25-hydroxyvitamin D3 and calcium supplementation. *Stroke* 1997; 28: 736-773.
- Takamoto S, Masuyama T, Nakajima M et al. Alterations of Bone Mineral Density of the Femurs in Hemiplegia. *Calcif Tissue Int* 1995; 56: 259-262.
- Demirbag D, Ozdemir F, Kokino S et al. The relationship between bone mineral and immobilization duration in hemiplegic limbs. *Ann Nucl Med* 2005; 19: 695-700.
- Bainbridge NJ, Davie MW, Haddaway MJ. Bone loss after stroke over 52 weeks at os calacis: influence of sex, mobility and relation to bone density at other sites. *Age Ageing* 2006; 35: 127-132.
- Hamdy RC, Moore SW, Cancellaro VA et al. Long-term effects of strokes on bone mass. *Am J Phys Med Rehabil* 1995; 74: 351-356.
- Yavuzer G, Ataman S, Suldur N et al. Bone mineral density in patients with stroke. *Int J Rehabil Res* 2002; 25: 235-239.
- Rammemark A, Nyberg L, Borssen B et al. Fractures after stroke. *Osteoporos Int* 1998; 8: 92-95.
- Pouwels S, Lalmohamed A, Leufkens B et al. Risk of hip/femur fractures after stroke. A population-based case-control study. *Stroke*; 40: 3281-3285.
- Kanis J, Oden A, Johnell O. Acute and long-term increase in fracture risk after hospitalization for stroke. *Stroke* 2001; 32: 702-706.
- Forster A, Young J. Incidence and consequences of falls due to stroke: a systematic inquiry. *BMJ* 1995; 311: 83-86.
- Pang MY, Eng JJ, Dawson AS et al. A community-based fitness and mobility exercise program for older adults with chronic stroke: a randomized, controlled trial. *J Am Geriatr Soc* 2005; 53: 1667-1674.
- Cheng PT, Wu SH, Liaw MY et al. Symmetrical body-weight distribution training in stroke patients and its effect on fall prevention. *Arch Phys Med Rehabil* 2001; 82: 1650-1654.
- Sato Y, Iwamoto J, Kanoko T et al. Risedronate therapy for prevention of hip fracture after stroke in elderly women. *Neurology* 2005; 64: 811-816.
- Poole KE, Loveridge N, Rose CM et al. A single infusion of zoledronate prevents bone loss after stroke. *Stroke* 2007; 38: 1519-1525.