

Osteoporosis Research: Assessment of Bone Density and Limb Function

John Delahunt, Department of Medicine, Wellington School of Medicine, Wellington, New Zealand

M. Elizabeth Falkner, MB, BS(Lond), LRCP (Lond), Medical Adviser, Post-Polio Support Society New Zealand, Inc., Masterton

THE RESEARCH

We all know that our polio-affected limbs are shorter and thinner than our unaffected ones. Most of us have seen x-rays showing that the affected bones are much thinner than the "normal" ones. Are they only smaller and thinner, but of typical strength? Or, are they actually weaker in structure, osteoporotic?

PRELIMINARY STUDY RESULTS

We studied 38 individuals who had residual weakness in one or more limbs after acute poliomyelitis. Polio onset was at a mean age of 53 years, (median 49, range 42-75). There were 11 men, mean age 61.3 years (60, 43-75) whose mean age of polio onset was 5.5 years, (1, 0-26). The 27 women had a mean age 62.9 years (range 41-85 years) at the time of the study. Four were premenopausal, 2 perimenopausal. The mean years since menopause of the 18 postmenopausal women was 17.7 years, (19, 1-34), mean age of polio onset of all women 10.7 years, (6, 1-37).

Six subjects developed polio after age 17, when they were fully grown. One man, now age 73, had polio at age 17. The mean age of the five women is now 70.7 (72, 62-73) years, onset mean age 30.4 (31, 24-37).

Weakness in individual limbs was self-assessed in a structured clinical interview on a 4-point grading system for the extensors and flexors of the upper and lower segments of

each limb for the current function, previous function during stable years, and at onset, where practicable.

Bone mineral content (BMC), lean tissue (LN), and bone area (BA) were measured in duplicate (with repositioning) in whole body scans for each subject using a fan beam Hologic QDR 4500 densitometer with regions of interest defined for whole limbs and parts of limbs.

Unilateral involvement occurred in 40 limbs (arms 18, legs 22), and bilateral in 16 limbs (arms 5, legs 11). *In all, there was a history of 47 fractures that occurred in 25 of the 38 cases.* Thirteen fractures were in long bones, of which 2 were at the lower end of the shaft of the femur (each in a polio-affected limb), 2 were at the femoral neck, with 5 tibial, 4 forearm, and 1 humeral.

For individual limbs BMC vs LN (dependent variable) the correlation coefficient (and regression estimate) was:

right arm (RA)	0.88	(15.4)
left arm (LA)	0.90	(16.8)
right leg (RL)	0.87	(18.2)
left leg (LL)	0.82	(18.1)

Thus for every unit (gm/cm²) increase of BMC in a limb, the estimate of lean tissue rose by 15-18 gms.

Associations of bone area vs. lean tissue:

right arm (RA)	0.80	(16.7)
left arm (LA)	0.82	(18.0)
right leg (RL)	0.84	(28.1)
left leg (LL)	0.70	(26.0)

The legs show a greater increase in lean tissue than the arms for any unit increase in projected bone area across the study population, with the results for all limbs pooled.

When comparing the arithmetic percentage difference in BMC and LN between abnormal and normal limbs for arms and legs separately, arms 0.75 (0.65), legs 0.85 (0.95), i.e., for every unit change in percentage difference in BMC between limbs there is a 0.65 gm percentage difference in LN in the arm and a 0.95 gm percentage difference in the leg. All correlations quoted were at the p=0.0001 level of significance.

CONCLUSIONS

Our data show that there is a strong association between the bone content (BMC) and lean tissue or muscle bulk (LN) in normal and atrophic limbs in post-polio individuals and that polio-affected limbs have a lower bone mineral content, in proportion to the reduction in muscle bulk. There also is an apparent association in the size of the limb (bone area, BA) and muscle bulk (LN), particularly in people whose polio developed at an early age. We had insufficient numbers of cases that developed polio after the age of 17 to allow us to analyze these effects independent of growth.

At this stage, we cannot say which of the two factors (muscle bulk or size of the limb) has the more important influence on bone content, but we suspect it is the muscle bulk. These results require further statistical analysis, particularly multivariate analysis of the factors that might predict BMC.

We suspect that the numbers of fractures, particularly the fractures of the femur, are more than would be expected in people of this age.

Adapted from *Assessment of Bone Density and Limb Functions in Subjects Who Have Had Acute Poliomyelitis in the Past* presented to the New Zealand Society of Endocrinology, Palmerston North (November, 1998) and to the Professor David Stewart Symposium, Dunedin (February, 1999). Other researchers involved in this project, in addition to polio survivor M. Elizabeth Falkner, include John W. Delahunt, Department of Medicine, Wellington School of Medicine; Robyn Green, Department of Public Health, Wellington School of Medicine; Jeremy Krebs, Capital Coast Health, Wellington Hospital; and Harry McNaughton, Department of Medicine, Wellington School of Medicine.

This study was not designed to assess fracture incidence and we cannot conclude this without a formal study (one is being planned).

If muscle bulk is subsequently shown to be more significant than limb development in the determination of limb BMC, a program of physical therapy seems unlikely to develop muscle bulk to the extent to reverse the loss of bone density, although it may minimize the amount of loss with age. Direct therapies to increase bone density, such as those being developed (for example, weight bearing during low-intensity vibrating stress), are likely to be more relevant. In fact, these may be more effective than the standard drug treatments for osteoporosis. The choice between a general drug treatment and a local limb physical treatment may (at least in part) depend on the general risk of osteoporosis, as assessed by standard DEXA scans of the hip and wrist in non-involved limbs.

WHAT DID OUR RESEARCH SHOW?

Yes, polio-affected limbs ARE weaker in actual structure; they are indeed relatively osteoporotic — even if the rest of our bones are not.

What about the progression of weakness? This is where the second stage of the research comes in — we have enough funds to repeat all the scans a year after the initial investigations. Several things will be interesting about this second step. We will repeat all the questionnaires on the degree of disability caused by polio both to show how reproducible people's accounts are to map deterioration over the year, to demonstrate any deterioration in bone density over the year, and to note any new fractures.

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WHAT OF POSSIBLE TREATMENT?

Everyone, men and women, would be well advised to have ordinary bone mineral density tests done. Men and pre-menopausal women can be affected, particularly if they get little weight-bearing exercise.

Anyone with generalized osteoporosis should definitely seek treatment, including a choice of many different drugs (not only hormone replacement) as well as lifestyle optimization.

What we do not know is whether ordinary medical bone-strengthening treatment will help people who have osteoporosis only in their polio-affected limbs. We speculate it will have only a limited effect. As there may be a relationship between the muscle or use of the limb and the bone content, it may be more beneficial to use local treatments. Weight-bearing on a vibrating surface is a physical technique being developed and that may be of interest to polio survivors.

If you have an ordinary bone density test (of lumbar spine and femoral neck) that is reported normal, it might be worth having a total body scan. Your physician can write a request to the radiologist to comment on osteoporosis in the individual limbs. However, this can be costly. After receiving the results, discuss with your doctor or an endocrinologist whether you should try a bone-strengthening drug for two years.

Also, then you can decide whether you want to take something that will help the osteoporosis in your polio limb. However, we do not know whether it would make any difference or not.

If you do decide to go down this path, we would be very pleased if you would send us copies of your bone density results and your treatment plan.

Please send the information to M. Elizabeth Falkner, MB, BS(Lond), LRCP(Lond), 132 Cole Street, Masterton, New Zealand. ■

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ISSUED QUARTERLY

International Polio Network (IPN)

4207 Lindell Boulevard, #110
Saint Louis, MO (Missouri)
63108-2915 USA
314-534-0475 ■ 314-534-5070 fax
Relay MO: 800-735-2966 TDD, 2466 V
gini_intl@msn.com, www.post-polio.org

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