Mobility status and bone density in cerebral palsy

S Wilmshurst, K Ward, J E Adams, C M Langton, M Z Mughal

Abstract

The spinal bone mineral density (SBMD) and calcaneal broadband ultrasound attenuation (BUA) was measured in 27 children with cerebral palsy. They were categorised into four mobility groups: mobile with an abnormal gait, mobile with assistance, non-mobile but weight bearing, non-mobile or weight bearing. Mean SD scores for BUA and SBMD differed among mobility groups (analysis of variance, \( p < 0.001 \) and \( p=0.078 \), respectively). (Arch Dis Child 1996;75:164–165)

Keywords: mobility, bone density, cerebral palsy.

Atraumatic fractures in children with cerebral palsy are known to be associated with low bone mineral density, which probably results from a combination of factors including immobility, low intake of minerals, and abnormal vitamin D metabolism in those treated with anticonvulsants. In this study, we measured the spinal bone mineral density (SBMD) and broadband ultrasound attenuation (BUA) of the calcaneum in children with cerebral palsy. We hypothesised that mobility impaired children with cerebral palsy would have reduced SBMD and BUA for age.

Subjects and methods

Twenty seven prepubertal white children (nine girls and 18 boys) with cerebral palsy aged 5 to 14 years attending schools for children with learning difficulties participated in the study. The study was approved by the Manchester research ethics committee. Written consent was obtained from the parents or guardians. Children were divided into the following mobility groups after discussion with parents and the child’s physiotherapist: group 1, mobile with an abnormal gait; group 2, mobile with assistance from a frame or rollator; group 3, non-mobile but regularly weight bearing in a frame; and group 4, non-mobile or weight bearing.

SBMD (g/ml) was measured using a rotating beam quantitative computed tomography (QCT) scanner (CT-9800; General Electric). Five millimetre sections were obtained through the midpoint of T12 to L3 vertebral bodies using a single energy (80 kV, 70 mA) technique. In vivo coefficient of variation for this technique is 3%. The BUA (dB/MHz) was measured in triplicate at the left calcaneum (a weight bearing bone consisting of 90% trabecular bone) of the children using a prototype paediatric contact ultrasound bone analyser (McCue Ultrasonics Limited) as previously described. In vitro and in vivo (in 6 to 18 year olds) coefficient of variation for BUA are 1.7% and 5%, respectively. BUA and SBMD values were transformed into z scores (SD scores) for age using the ‘in-house’ BUA data from 399 healthy children from Manchester and Sheffield and published SBMD values in children from Los Angeles, USA.

Simple linear regression analysis was used to compare BUA with SBMD, and one way analysis of variance was used to compare the differences in the mean BUA and SBMD z scores in different mobility groups.

Results

The mean (SD) age was 9.2 (2.4) years. Ten children were excluded from the analysis of the SBMD data because of vertebral abnormalities and poor scan quality caused by movement. As shown in fig 1, BUA was significantly related to SBMD (\( r=0.60, \ p=0.01 \)). The mean BUA z scores were significantly different (\( F=8.958, \ p < 0.001 \)) among children in the four mobility groups (table 1). The same trend was seen for SBMD (table 1) but the difference did not reach statistical significance (\( F=2.851, \ p=0.078 \)).

Discussion

Our data confirms the findings of Shaw et al of reduced SBMD in severely disabled children with cerebral palsy. Bone strength and fracture risk in osteoporotic patients is however related to both the reduction in bone mineral density and disruption of microstructural parameters (trabecular number, thickness and connectivity), which cannot be assessed by bone densitometry. In this study we therefore also measured the calcaneal BUA, which is related to the structural features of the bone as well as its density. Our results show that BUA is reduced for age in children with cerebral palsy.
Table 1  Mean (SEM) z scores for the four mobility groups

<table>
<thead>
<tr>
<th>Mobility Group</th>
<th>BUA (dB/MHz)</th>
<th>SBMD (g/cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1 (mobile with an abnormal gait)</td>
<td>11 - 1.07(0.30)</td>
<td>7 - 1.08(0.32)</td>
</tr>
<tr>
<td>Group 2 (mobile with a frame or rollator)</td>
<td>4 - 1.85(0.51)</td>
<td>4 - 2.12(0.59)</td>
</tr>
<tr>
<td>Group 3 (non-mobile but stands in a frame)</td>
<td>6 - 2.46(0.15)</td>
<td>3 - 1.45(0.38)</td>
</tr>
<tr>
<td>Group 4 (non-mobile or weight bearing)</td>
<td>6 - 3.09(0.24)</td>
<td>3 - 2.79(0.47)</td>
</tr>
</tbody>
</table>

One way analysis of variance: BUA, F=8.958, p < 0.001; SBMD, F=2.851, p=0.078.

We also found that the degree or reduction in BUA measured as deviation from the mean for age (z score) was associated with the degree of immobility and non-weight bearing. The similar relationship between SBMD z scores and mobility status was weak. This may have been due to the smaller numbers in the SBMD group owing to exclusion of children with poor quality QCT scans. An alternative explanation, especially in those who were wheelchair bound, may be that in these children there was weight loading on the spine but little or none on the lower limbs and thus on the calcaneum, the site of BUA measurement. To the best of our knowledge, this is the first study to compare SBMD measured by QCT to BUA in children. The less than perfect relationship between these two techniques of bone density measurement (r=0.60, p=0.01) may be due to different bone density being found at the skeletal sites and/or because BUA also reflects properties other than bone density. 5

The implication of results of this study is that weight bearing exercise programs might help to moderate bone loss in mobility impaired children with cerebral palsy. An intervention study might be required to establish if the observed relationships are in fact causal. Bisphosphonates, which act as inhibitors of osteoclast mediated bone resorption, might offer another approach to treatment of osteoporosis in children with cerebral palsy. 2 However, these products are not licensed for children and therefore their use is experimental at present.

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