CLINICAL RESEARCH

Bone mineral content in Polynesian and white New Zealand women

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Abstract

The forearm bone mineral content of Polynesian and European women in New Zealand was measured to assess whether the inter-racial differences found in other populations also occurred in these two groups. The bone mineral content of the non-dominant distal radius and ulna was measured by single photon absorptiometry in 123 European and 80 Polynesian women. The mean values were about 20% higher in Polynesians than in Europeans.

The reason for this difference in bone mineral content is unknown but the findings do show that high bone density is not confined to African races and that inter-racial differences in bone mineral content may be more common than has been thought hitherto.

Introduction

Race is an important determinant of bone density and thus of the incidence of osteoporosis. Studies have assessed bone mass in peoples of European, African, and Oriental origin but have neglected the indigenous peoples of the Pacific and the remainder of Asia. New Zealand has a large Polynesian population comprised of the indigenous Maoris and of immigrants from the south west Pacific. We studied the forearm bone mineral content of these groups of adult New Zealand women and compared it with that of white New Zealand women.

Patients and methods

Women aged 18-70 years were recruited from hospital staff and visitors, mothers taking their children to a local medical centre, and church congregations. Individuals with any major systemic illness or a history of glucocorticoid or postmenopausal oestrogen therapy and those who had undergone bilateral oophorectomy were excluded. Fifty Maori and 30 other Polynesian women, aged 18-66 years, and 123 European women, aged 18-69 years, met these criteria and were studied. The bone mineral content of the non-dominant distal radius and ulna was measured using a Novo GT 35 osteodensitometer with an iodine-125 source.[1]

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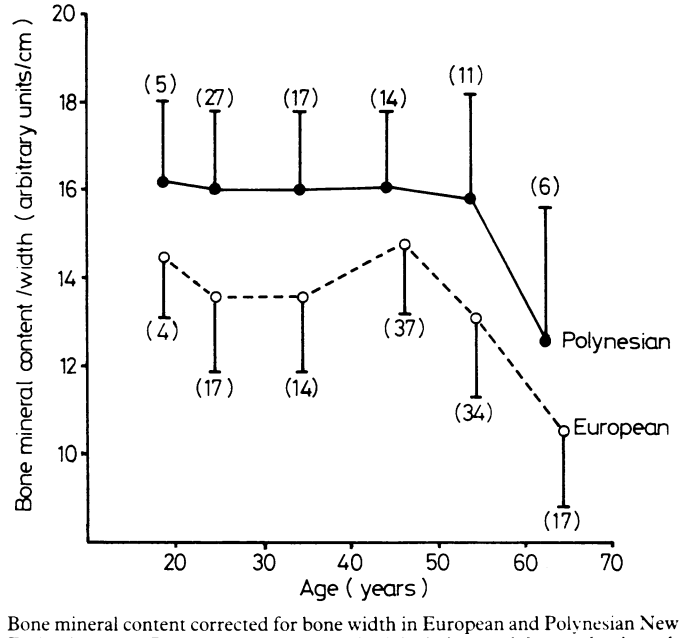
Results

There were no significant differences in bone mineral content between Maori and other Polynesian women. The results in these two groups were therefore pooled for all subsequent analyses. Bone mineral content was significantly higher in Polynesian women than in Europeans, whether tested by parametric or non-parametric analysis of covariance with age as the covariable ($p<0.0001$; see figure). The mean values were about 20% higher in Polynesians.



Bone mineral content corrected for bone width in European and Polynesian New Zealand women. Data are means and standard deviations and the number in each group is indicated in parentheses. The two groups were significantly different ($p<0.0001$).

Bone density showed a significant decline with age in both races ($p<0.0001$). The mean age at menopause did not differ between the two groups (Polynesians 48·2 (SEM 0·9) years, Europeans 49·6 (0·5) years).

Discussion

These data show that the bone mineral content of the distal forearm is higher in Polynesian women than in New Zealand women of European ancestry. This finding is consistent with the previously documented low incidence of femoral neck fractures in this group.[2]

The reasons for inter-racial differences in bone mass remain uncertain. While nutrition and lifestyle probably contribute in some instances, they are unlikely to be relevant to the present findings as both groups live in the same homogeneous society. In American blacks an increase in muscle mass has been shown[3] and has been suggested to be causally related to their higher bone density. The increased muscle mass, however, could also be regarded as an independent manifestation of a generalised increase in connective tissue mass. Recently differences in serum concentrations of parathyroid hormone and vitamin D metabolites have been found between American blacks and whites.[4] This may imply that race has a major effect on the control of calcium metabolism or it may merely be a reflection of reduced cutaneous vitamin D synthesis secondary to dark skin colour. The former possibility is supported by the independent finding of higher calcitonin and katacalcin levels in blacks.[5]

These findings indicate that high bone density is not unique to African races and that inter-racial differences in bone mass may be more common than was once thought.

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Morphine intoxication in renal failure: the role of morphine-6-glucuronide

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Abstract

Patients with impaired renal function may experience severe and prolonged respiratory depression when treated with morphine. This has been attributed to accumulation of the drug during renal failure. Three patients are described who had classical signs of intoxication with morphine in the absence of measurable quantities of morphine in the plasma. The observed clinical effect is attributed to accumulation of the pharmacologically active metabolite morphine-6-glucuronide, which is usually renally excreted. It is concluded that morphine does not accumulate in patients with renal failure but that accumulation of metabolites does occur.

The previously reported observations of morphine accumulation during renal failure probably result from the use of radioimmunoassays that cannot distinguish between morphine and morphine-6-glucuronide. Thus the apparent morphine concentration measured with these assays in fact reflects the total quantity of morphine and morphine-6-glucuronide present.

Introduction

Patients with impaired renal function may experience severe and prolonged respiratory depression when treated with morphine. This sensitivity to morphine has been attributed to accumulation of the drug due to decreased metabolism or elimination.[1]

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We report on the morphine and metabolite concentrations of three patients with renal failure who experienced pronounced respiratory depression apparently caused by treatment with morphine.

Patients, methods, and results

Case 1—A 64 year old man with chronic renal failure (creatinine clearance 3 ml/min) underwent elective aortic aneurysm repair. Postoperatively he received 84 mg of Omnopon in 42 hours. (Omnopon comprises morphine and noscapine with small quantities of codeine and papaverine. Noscapine and papaverine have no analgesic or respiratory depressive effects.) Three days postoperatively, he developed respiratory depression, which necessitated mechanical ventilation, and treatment with Omnopon was stopped. No other respiratory depressant drugs had been given. Naloxone was given to maintain spontaneous ventilation, and 20·3 mg was required over the next eight days.

Case 2—A 39 year old woman with polycystic kidneys (plasma creatinine concentration >1000 μmol/l (11 mg/100 ml) required ventilation after a subarachnoid haemorrhage. She received 361 mg Omnopon over five days. Respiratory depression requiring ventilation and reversible by naloxone persisted for six days after treatment with Omnopon was stopped.

Case 3—A 70 year old man underwent emergency surgery for peritonitis and subsequently developed severe acute renal failure. He received 415 mg Omnopon over three days. Respiratory depression requiring ventilation persisted for three days after treatment with Omnopon was stopped.

During the long period of respiratory depression apparently induced by morphine in these patients plasma samples were collected and analysed using a high performance liquid chromatography assay.[2] This assay distinguishes and measures morphine and its major metabolites, morphine-3-glucuronide, morphine-6-glucuronide, and normorphine.[3] The table shows the concentrations of morphine, morphine-6-glucuronide, and morphine-3-glucuronide. (No normorphine was detected in any sample.)

Discussion

We have described three patients with prolonged respiratory depression after treatment with morphine in the presence of