

Adult Nutritional Osteomalacia in Black Patients

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SUMMARY

Three patients suffering from nutritional osteomalacia are described. Nutritional osteomalacia is rare in Southern Africa, in spite of diets with low vitamin D content, because of the high solar irradiation of the skin. All 3 patients responded biochemically and radiologically to small doses of vitamin D. In 1 patient the radiological changes of secondary hyperparathyroidism are described; this feature is uncommon in nutritional osteomalacia. The possible reasons for nutritional osteomalacia in Blacks are discussed.

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Nutritional osteomalacia in Blacks is rare. We could find no published report of this entity in Blacks in the South African literature, and only 1 other case of nutritional osteomalacia in a White spinster, who had a personality defect and possibly did not expose herself to sunlight, has been described by Weinkove *et al.*¹ We record 3 patients suffering from nutritional osteomalacia seen over a period of 3 years.

CASE REPORTS

Case 1

A 30-year-old female was admitted with a 6-month history of severe pain in both lower limbs, backache and the inability to walk. Her diet consisted of mealie meal, cabbage, but no dairy products, fish or meat. On examination she was found to be short in stature, with bowed lower limbs and unable to stand unless supported. She was irritable and unco-operative with generalised scabies. Special investigations included the following relevant findings: haemoglobin 12,8 g, erythrocyte sedimentation rate (ESR) 24 mm/hour (Westergren), leucocyte count 9 000/mm³, serum calcium 8,5 mg/100 ml, serum phosphorus 2,4 mg/100 ml, serum alkaline phosphatase 25 KA units (normal 1-4), serum albumin 4,2 g/100 ml. A renal cause for the osteomalacia and malabsorption was excluded. X-ray films of the skeletal system showed generalised loss of bone density; the pelvis had a triradiate appearance with protrusion of the head of the femur into the acetabulum (Fig. 1); there were pseudofractures in the bones of the extremities, superior and inferior pubic rami, ribs, scapulae and the 2nd and 3rd right metacarpal

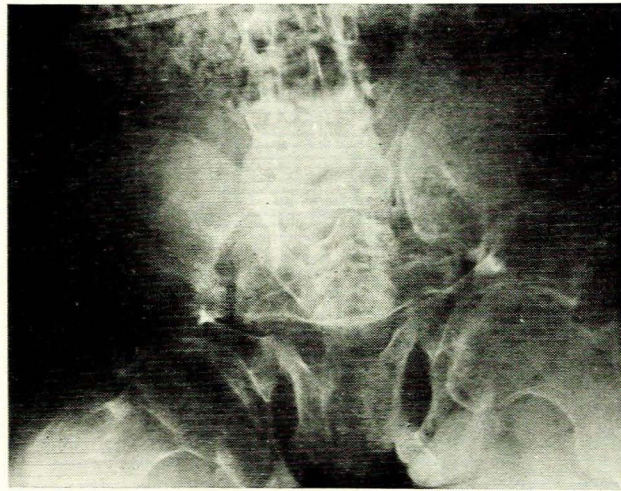


Fig. 1. X-ray film of pelvis showing radiolucency of the bones, triradiate pelvis and protrusion of the head of the femur into the acetabulum.

bones; and loss of lamina dura of the teeth. X-ray films of the abdomen excluded nephrocalcinosis.

The patient was treated with vitamin D 4000 units orally daily. Clinically she improved 2 weeks after therapy, and biochemical investigations 4 months after therapy showed a serum calcium of 9 mg/100 ml, serum phosphorus 3,2 mg/100 ml, and serum alkaline phosphatase 40 KA units. The phosphorus excretion index was normal. A skeletal X-ray survey showed healing of the pseudofractures and recalcification of the bones. Clinically she was free from pain and able to walk.

Case 2

A male aged 17 years was admitted with a long-standing history of pain in the limbs, aggravated by movement, and weakness of the lower limbs; he was able to walk only with the support of a stick. There was no family history of osteomalacia. His diet consisted of mealie meal and cabbage, with no meat or dairy products. Physical examination revealed bilateral gynaecomastia but no evidence of malnutrition; all other systems were normal. Routine and renal special investigations were normal except for an unexplained eosinophilia of 23%. Serum calcium was 9 mg/100 ml, serum phosphorus 1,8 mg/100 ml, serum alkaline phosphatase 23 KA units, serum albumin 4,6 g/100 ml, serum gamma globulin 1,2 g/100 ml, and phosphorus excretion index +0,1. Intravenous pyelography showed clubbing of the calyces suggestive of pyelonephritis; a micturating cystogram was normal. A renal biopsy showed evidence of focal pyelonephritis. The stools were negative

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for parasitic infestation. Malabsorption syndrome and muscle dystrophy were excluded. X-ray films of the skeletal system showed generalised rarefaction of the bones, numerous cystic lesions in both heads of the femur, loss of the lamina dura of the teeth and pseudofractures in the shafts of both femurs. The vertebral bodies showed increased density in both superior and inferior aspects (rugby jersey appearance). The hands showed marked subperiosteal and subepiphyseal erosions (Fig. 2), indicating secondary hyperparathyroidism.

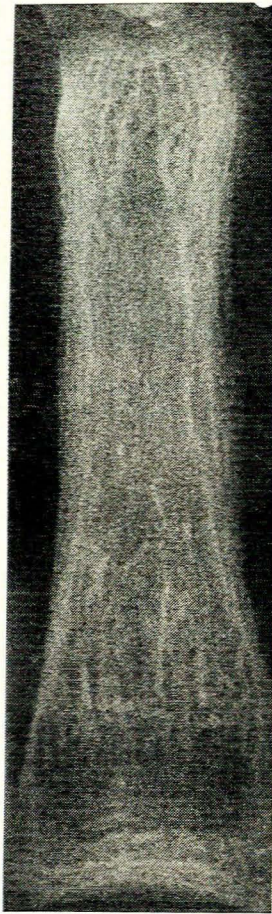


Fig. 2. X-ray film of phalanx with marked subperiosteal erosion indicating secondary hyperparathyroidism.

The patient was treated with vitamin D 500 units daily, and after 2 months of therapy the biochemical investigations were: serum calcium 9,4 mg/100 ml, serum phosphorus 3,0 mg/100 ml and serum alkaline phosphatase 59 KA units. Clinically the patient improved and he was able to walk unaided. A skeletal survey showed healing of the pseudofractures, remineralisation of the bones and resolution of the subperiosteal erosions in the hands.

Case 3

A male aged 36 years was admitted in a mentally confused condition, after he had had generalised convulsions for one month; he became progressively weaker until he was unable to stand or walk. He was not on any anti-epileptic therapy to account for his osteomalacia. The patient had been mentally retarded for a long time and unable to care for himself. His diet had consisted of mealie meal and vegetables, with no dairy products or meat. There was no family history of epilepsy or osteomalacia. He spent most of his time indoors.

Physical examination showed a malnourished, irritable and confused patient. His limbs were rigid and he had dependent oedema. A positive Chvostek and Trousseau's sign was present. Central nervous system examination showed a confused patient with hyperreflexia and rigidity of the limbs. The other systems were normal. Special investigation findings included haemoglobin 11,4 g/100 ml, leucocyte count 12 000/mm³ ESR 46 mm/hour; serum calcium 4,4 mg/100 ml, serum phosphorus 1,6 mg/100 ml, serum alkaline phosphatase 26 KA units, serum albumin 2,7 g/100 ml, and a phosphorus excretion index of +0,13. A renal or malabsorption cause for the osteomalacia was excluded. An electrocardiogram showed a corrected Q-T interval of 0,4 seconds. A skeletal survey showed that there were fractured ribs on the right side of the chest, pseudofractures in both scapulae and erosions of the lateral ends of the clavicles. There was generalised radiolucency of the bones and loss of the lamina dura of the teeth.

The patient was treated with vitamin D 4 000 units orally, with improvement within a week. After 4 months the serum calcium was 8,3 mg/100 ml, serum phosphorus 5,6 mg/100 ml, serum alkaline phosphatase 36 KA units and serum albumin 4,1 g/100 ml. Within a period of 6 months there was radiological healing of the pseudofractures, fractured ribs, reappearance of the lamina dura in the teeth and remineralisation of the bones.

DISCUSSION

Thomson³ showed that a large proportion of ultraviolet light of a wavelength which included the antirachitic range (290 - 320 nm) could easily penetrate the stratum corneum of white skin. A dark skin has been widely supposed to lessen transmission of ultraviolet light into the deeper layers of the epidermis and thereby reduce the skin synthesis of cholecalciferol. Stanbury⁴ feels that there is no valid reason why darkly pigmented skin should impede the formation of vitamin D. However, Stamp⁵ states that it is almost certain that dietary sources of vitamin D are only required when a person is shielded from effective sunshine by clothing, housing conditions or industrial smog.

The criteria in the diagnosis of nutritional osteomalacia in our patients were negative investigations and past history for steatorrhoea; a diet grossly inadequate in vitamins; a good clinical and biochemical response to small doses of oral vitamin D, and negative investigations for renal disease.

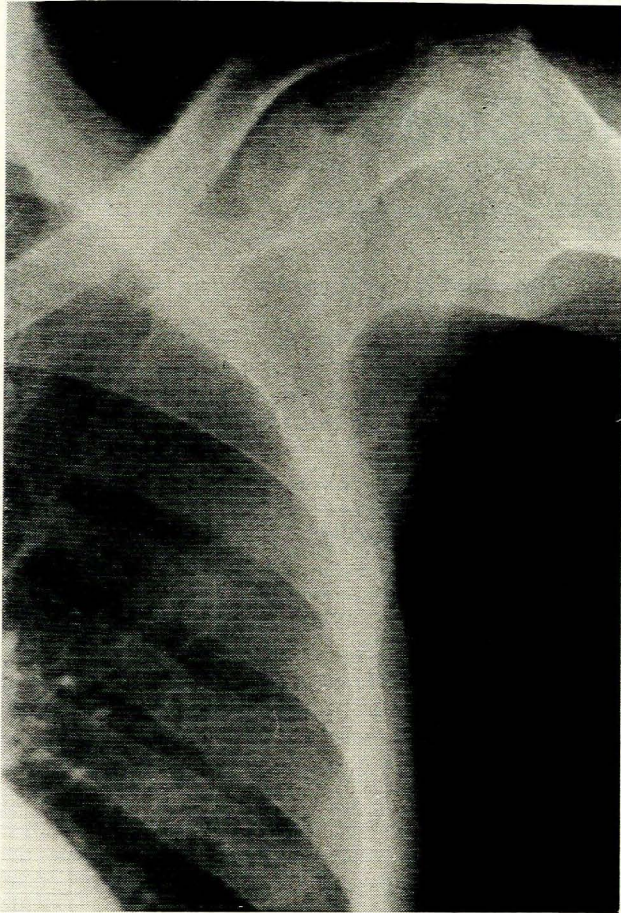


Fig. 3. Radiological evidence of pseudofracture (Looser's zone)—evidence of osteomalacia.

In case 2 evidence of pyelonephritis was demonstrated but it is unlikely that it caused the osteomalacia, since the blood urea was normal and a tubular defect was excluded. Moreover this patient responded biochemically and radiologically to small doses of vitamin D. All 3 patients had a poor dietary history of vitamin D. In case 3, because he was an epileptic and mentally defective, it is possible that lack of solar irradiation, because he led an indoor life, contributed to the osteomalacia. However, in cases 1

and 2 there was no evidence of a lack of irradiation, and it may be that lack of penetration of sunlight into a pigmented skin with associated deficiency of dietary vitamin D could have been responsible for the osteomalacia.

Radiological evidence of secondary hyperparathyroidism is extremely uncommon. Dancaster and Jackson⁶ did not find this entity in 54 Black children suffering from rickets. Radiological evidence of secondary hyperparathyroidism in nutritional rickets has been described in a 7-year-old idiot.⁷ In case 2 evidence of secondary hyperparathyroidism in nutritional rickets was present (Fig. 3). However, while radiological evidence of secondary hyperparathyroidism in nutritional rickets is very uncommon, other parameters of hyperparathyroidism in nutritional rickets have been described: firstly, that the phosphorus excretion index was found to be elevated in vitamin D deficiency rickets.⁸ All our patients had an increased phosphorus excretion index. Secondly, elevated concentrations of immunoreactive parathyroid hormone were found in 7 out of 10 children with nutritional rickets.⁹ We feel that the reason why radiological changes of secondary hyperparathyroidism occurred in our case was probably because the nutritional osteomalacia was of long duration.

This article records 3 rare cases of nutritional osteomalacia which in cases 1 and 2 were probably due to the lack of dietary vitamin D, combined with lack of penetration of ultraviolet light into the stratum corneum of a pigmented skin. In case 3 the osteomalacia was probably due to the lack of dietary vitamin D, together with lack of solar irradiation because the patient led an indoor life. This article also illustrates that nutritional osteomalacia may rarely occur, in pigmented patients if their diet lacks vitamin D. This entity in our experience is very rare, and in general it accords with earlier epidemiological work done on the Indian subcontinent and that recently recorded by Hodgkin *et al.*¹⁰ that vitamin D deficiency is seldom found in areas with high solar irradiation.

REFERENCES

1. Weinkove, C., Pimstone, B. L. and Jackson, W. P. U. (1971): *S. Afr. Med. J.*, **45**, 81.
2. Wrong, O. and Davies, H. E. T. (1959): *Quart. J. Med.*, **28**, 259.
3. Thomson, M. L. (1955): *J. Physiol. (Lond.)*, **127**, 236.
4. Stanbury, S. W. (1973): *Lancet*, **2**, 446.
5. Stamp, T. C. B. (1973): *Arch. Dis. Childh.*, **48**, 2.
6. Dancaster, C. P. and Jackson, W. P. U. (1962): *S. Afr. Med. J.*, **36**, 364.
7. Franconi, G. (1960): *Ibid.*, **34**, 769.
8. Taitz, L. S. and De Lacy, C. D. (1962): *Paediatrics*, **30**, 875.
9. Joffe, B. I., Hackeng, H. L., Seftel, H. C. and Hartdegen, R. G. (1972): *Clin. Sci.*, **42**, 113.
10. Hodgkin, P., Kay, G. H., Hine, P. M., Lumb, G. A. and Stanbury, S. W. (1973): *Lancet*, **2**, 167.