OSTEOMALACIA

MOHAMMAD ALI KHAN, S MUSANIF SHAH, MASOOD UR REHMAN, MOHAMMAD ILYAS SAEEDI AND MOHAN KUMAR

Department of Medicine,
Postgraduate Medical Institute,
Lady Reading Hospital, Peshawar.

INTRODUCTION

Osteomalacia and rickets result from defective mineralization of bone which is usually caused by insufficiency of vitamin "D". Osteo-malacia refers to clinical consequences of defective mineralization of the organic matrix of adult skeleton.[1] The term rickets is reserved for the same pathology in growing skeleton. It is mostly caused by vit. "D" insufficiency, however defective mineralization can also be due to resistance to vit. "D", hereditary hypophosphatemia, hypophosphatasia and certain renal tubular disorders.[2,3,4] Major dietary sources of vitamin "D". Are fortified foods, fish oils and dairy products. Poor diet, multiple pregnancies and lactation are the main causes of osteomalacia in our patients. Common presenting features are bone pains and deformities of the skeletal system. Every year our unit diagnose on an average 3 cases of Osteomalacia. Typically the patient is a multiparous female, with poor dietary history. Occasional cases of malabsorption and chronic renal failure causing osteomalacia are also noted. In this paper we report a young unmarried girl with osteomalacia due to poor diet.

CASE REPORT

A sixteen year old Afghan girl, was brought on 13.05.1993 to Medical C Unit with generalized aches and pains and progressive weakness of the lower limbs for the last three months. She was treated locally but did not improve. There was no history of any chronic illnesses and her bowels were regular. She lost her mother at the age of seven years and after that she was being looked after by her step-mother. Dietary history revealed that her diet consisted mainly of wheat bread and black tea. Dairy products and meat were very seldom available to her. She used to do her household work and had sufficient exposure to sun.

On examination she looked pale, was lying listlessly and could not move her lower limbs. There was generalized wasting of muscles and legs were bowed. The power in the upper limbs was of grade 4/5 while in the lower limbs it was 2/5. Reflexes were normal. Joints of the extremities were normal. Both active and passive movements were restricted. Tenderness could be elicited on bony prominences. Rest of the systemic examination was unremarkable. The following investigations were carried out and reported as within normal limits. Haemogram, serum urea, creatinine, CPK, Albumin, L.F.T, Xylose absorption test, stool test and abdominal ultrasound. Skeletal survey showed Looser's Zones in the femur, pubic rami and scapulae. (Fig. 1) S. Calcium was

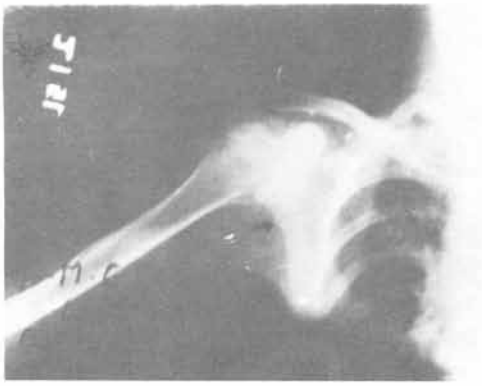

Fig. 1.

low, 7.5 mg/dl (N. 9–11 mg/dl) and alkaline phosphatase was high, 454 u/l (N upto 329 u/l).

Osteomalacia was the most likely diagnosis in the light of the above investigations and to confirm it bone biopsy was performed. The result was suggestive of osteomalacia. After this to determine the cause tests for malabsorption and urinary phosphates were performed which turned out to be normal. So nutritional osteo-malacia was the final diagnosis which was supported by the patients dietary history and absence of any obvious secondary cause. She gave a dramatic response to intensive oral vitamin "D" and calcium therapy.

The patient was given the following treatment:-

1. Alpha Calcidol Capsule 0.25 Microgram 1 TDS.
2. Tab Caltract 600 (calcium + vit. D preparation)
3. Physiotherapy.

There was marked improvement in her condition and she was able to walk with the help of support in two weeks time. Her pains also disappeared.

DISCUSSION

Osteomalacia and rickets result from a defect in the mineralization of bone, which is usually caused by insufficiency of vitamin "D". The main source of vitamin "D" is endogenous, that is the ultraviolet irradiation of 7-dehydrocholesterol in skin. Major dietary sources are fortified foods, fish oils and dairy products. To become active, vitamin "D" undergoes two stage of hydroxylation: first in the liver, forming 25-hydroxy vitamin "D" and then in the kidney, making 1 - 25 dihydroxy vitamin "D" (1 - 25 $(OH)_2$ D). This is the most active metabolite of vitamin "D". As the hydroxylation takes place in the liver and kidney, chronic diseases of these organs interfere with vitamin "D" metabolism and can cause osteomalacia.[5,6]

Osteomalacia may present in different ways. The prominent feature may be bone pains leading to an incorrect impression of arthritis. Osteomalacia may incorrectly be diagnosed as bone fractures. Proximal myopathy may be prominent feature leading to futile investigation for primary muscle diseases, neurological diseases and neoplasm.

Nutritional osteomalacia usually results from a combination of inadequate exposure to sun light and a diet poor in vitamin "D". This usually occurs in elderly house bound individuals and infants. Coeliac disease and other malabsorption syndromes may lead to vitamin "D" deficiency.

In our patient the only positive history was that of a deficient diet. She was not totally house bound but her diet was almost devoid of vitamin "D". She was living on wheat-bread and tea. Phytates in wheat interfere with calcium absorption and may aggravate the condition. There was no evidence of malabsorption syndrome, chronic liver or renal disease. Moreover her response to oral vit. "D" therapy suggests vit "D" insufficiently in the diet as cause of her osteomalacia.

There are some other conditions to be considered before making a firm diagnosis of pure nutritional vitamin "D" deficiency. In familial hypophosphataemic rickets (Vitamin "D" resistant rickets, phosphaturic rickets) the usual age of presentation is 1-3 years. There is a defect in the tubular reabsorption of phosphate. In a few cases renal glycosuria has been reported but the other tubular functions remain normal.[7,8] In adults the picture is that of severe osteoporosis with pseudo fractures (Looser's Zones) sometimes called the milkman syndrome. Urinary excretion of phosphate is raised. The condition needs much higher doses of vitamin "D", 50,000 -100,000 units daily. High doses of sodium phosphate are used orally to bring the serum phosphate concentration to normal.

In hypophosphatasia, which is a disease of the bone due to a rare inborn error of metabolism, the clinical and radiological picture may be similar to osteomalacia and rickets depending upon the age of patient and severity of the disease. However, the bio-chemical abnormalities clearly differentiate it from osteomalacia and rickets. In hypophasphatasia plasma alkaline phosphatase is low, plasma calcium normal or raised and plasma phosphorous is low. In hyposphatasia vitamin "D" is contraindicated because it will induce severe hypercalcemia.

In our patient the urine and blood pH were normal which rules out renal tubular acidosis. She was not on anticonvulsant drugs which could have caused vitamin D metabolism disturbance and osteomalacia.

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