A case of rickets in a child on prolonged phenobarbitone therapy

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Introduction

Rickets is the clinical manifestation of failure in mineralization of growing bone. Failure of mature bone to mineralize is called osteomalacia. Rickets and osteomalacia are recognized complications of long term anti-epileptic medications¹²³⁴. Anticonvulsants exert their deleterious effects on mineral and bone metabolism by inducing hepatic microsomal enzyme systems, resulting in increased catabolism and excretion of vitamin D and its biologically active products⁵. Though all commonly used anticonvulsants have the propensity to cause rickets, phenytoin and phenobarbitone have the greatest potential⁵⁶.

Case report

A two years and ten month old boy had been on phenobarbitone therapy since the age of 2½ months, for convulsions following birth asphyxia. It was noted during clinic visits, that the child was developing bow legs and widening of wrist joints. X-rays of wrists confirmed changes of rickets (Figure 1). His alkaline phosphatase was 23 k.a.u. He was started on 1-alpha therapy and phenobarbitone was replaced with sodium valproate. His clinical symptoms of rickets improved with the above management and subsequent x-rays showed healing ends of long bones (Figure 2). The height and weight parameters of the child fell between the 10th and 25th centiles for age. He had no clinical evidence of malnutrition. The child was adequately exposed to sunlight.

The presence of adequate exposure to sunlight makes a diagnosis of nutritional rickets unlikely but the possibility cannot be completely excluded. Epileptic children on long term anticonvulsants should be closely supervised for the development of rickets. Biochemical changes are more common than overt rickets. Since the treatment is easy and effective, the need for early diagnosis cannot be over emphasized.

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References


