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ROLE OF VITAMIN D ON RICKETS

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ABSTRACT

Vitamin D has traditionally been viewed as a fundamental hormone in the regulation of phosphorus and calcium and bone metabolism. In recent years, the discovery of a new world of extra skeletal and particularly immune modulator effects renewed the interest of research on vitamin D. In the present experiment we are studying the role of vitamin d in rickets

Keywords: Vitamin D , Rickets

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INTRODUCTION

The long bones develop embryologically on a cartilaginous model and hence are called cartilaginous bones although most, of the diaphysis is ultimately replaced by periosteal (membranous) bone. Most of the increase in length of the long bone is brought about by interstitial growth. The increase in diameter of both the shaft and its marrow is due to periosteal deposition of new bone and endosteal resorption of older bone. In the epiphyseal cartilage and the adjoining region of diaphysis (metaphysis), 5 zones can be normally identified. 1. Resting cells, some of which by division lead to (.2) the columns of proliferating cells, (3) each cell of which develops into a cylinder thereby compressing and elongating the surrounding matrix. It is in zone 2 and 3 that the increase in length of the bone occurs. (4) The matrix, primarily the longitudinal trabeculae,than calcifies with hypertrophy, vacuolization and death of the cells. 5. The uncalcified, or less well calcified transverse cartilaginous traheculae are destroyed by marrow elements. 33 Osteoblasts which develop from the connective tissue cells of the marrow penetrate between the calcified longitudinal trabeculae and deposit the bone matrix on the exposed surface. The bone matrix is promptly calcified producing a firm union between the cartilaginous and osseous portions of the bone. The characteristic features of rachitic metaphyseal region are (i) a near normal rate of growth and proliferation of zone 2 and 3, (ii) lack of mineralization and maturation of the cartilagenous matrix and cells (zone 4) and (iii) reduced erosion and uneven penetration by elements from the marrow (zone 5) and renewed but atypical removal of the cartilage leading to a mixed mass of cartilage and osteoid. Even short term vitamin D deprivation in pregnant rats (6th day of pregnancy onward) leads to the characteristic changes in the long bone of the pups. Boass et al. (19.81a) observed the changes described above in 19 dayjold pups. Rickets and Osteomalacia can be produced in experimental animals. Initially rickets was determined by gross examination of the width of epiphyseal plate in the long bones and the increased width was easier to distinguish when the diet was low in vitamin D as well as phosphorus.This led to the belief that low serum phosphorus is necessary in the rat to cause rickets and low serum calcium due to vitamin D deficiency alone does not cause rickets. 34 However such rats did not gain weight; their growth was stunted and the rats soon died presumably as the result of malnutrition. Therefore commonly used vitamin D deficient diet contained only 0.44% calcium and 0.3% phosphorus so as to produce hypocalcemia as well as hypophosphatemia. Rasmussen (1969) fed rats on vitamin D deficient diet containing 1.1% calcium' and 0.8% phosphorus for eight weeks. Such animals showed widening of epiphyseal plate in the tibia indicating that rickets can be induced to develop in vitamin D deficient rats despite normal serum phosphorus. Rickets is not necessarily associated with vitamin D deficiency alone. Rats fed on low phosphorus diet adequate in vitamin D

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also develop rickets (Carttar et al.,1950). Rickets has also been reported in the rat on low calcium'diet (Shohl, 1936). However calcium deficiency also increases parathormone secretion resultin 35 age, in the stage of active growth,, had 25(OH)D and l,25(OH)2D levels undetectable or very low. Tibial epiphyseal plate was normal in the rat with normal serum calcium and slightly low serum phosphorus but widened in the rat with low serum calcium as well as serum phosphorus. Numerous studies on weaning rats have shown that rickets, evidenced by widened epiphyseal cartilage and hypophosphatemia can be produced only by depriving the animals of vitamin D as well as phosphorus for several weeks (Boass et al.,1981). However typical rickets may be observed in pups by 20 days of age when the mothers are deprived of only vitamin D from 6th day of pregnancy. Boass et al (19810? have shown that with such a procedure pups at 8th day of age had hypocalcemia and hypophosphatemia. By 15th day of age serum 25(OH)D was undetectable, serum calcium and phosphorus were low and body- weight was reduced by 26%. In 19 days old pups, the ratio of bone weight to body weight was not reduced but ash weight as a percent of bone weight was 33.8% an compared to 36.2% in control pups. Histological examination of the tibia revealed the characteristic feature of rickets including irregularity and widening of the hypertrophic cartilagecell layer, trneven tine of ossification and widened and irregularly arranged bone trabeculae. Thus rat pups suckling vitamin D deprived mothers can develop biochemical and histological evidence of vitamin D deficiency similar to that of human vitamin D-deficiency rickets.

Vitamin D and its metabolites circulate in the blood bound to a specific transfer protein called vitamin D-binding protein (DBP). DBP expresses binding preference for 25(OH)D.j, 24,2 5 (OH and 2 5,2 6 (OH ^D^ when compared to the parent vitamin and l,25(OH)2D2 (Haddad and Walgate, 1976; Imawari et al., 1976; Belsey et al.,1974). In man, DBP appears to be an alpha globulin with a molecular weight of 60,000, In human plasma DBP concentration is 525 ug/ml in normal individuals but reaches upto 1,254 ug/ml in pregnant women. Since the total amount of vitamin D sterols in normal individuals is approximately 35ng/ml (Lambert et al.,1981; Shepard et al.,1979), it has been calculated that under normal circumstances 98% of the plasma DBP circulates with its binding sites unoccupied by any vitamin D metabolite. Human milk contains two types of DBPs, one which appears to be identical to the plasma DBP while the other 31 resembles DBP previously isolated from a number of different tissues (Vanbaelen et al.,1977), Actual level of DBP in human milk seems to be rather low. In early lactation it is 18 ug/ml but 3 weeks after initiation of lactation, it is about 3 ug/ml i.e. about 1.2% of the plasma DBP level of a normal woman (Haddad and Walgate, 1976). Maternal blood levels of various vitamin D metabolites determine the amount of these metabolites in the milk. Milk from a mother who is vitamin D-deficient would be'devoid of vitamin D and its metabolites. On the other hand, vitamin D content of milk can be increased

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by administration of a large amount of vitamin D to the mother (Polskin et al.,1945; Hibb and Ponden, 1955). Like plasma the concentration of 25(OH)D seems to be the stable in the milk, whereas concentration of the parent vitamin is variable depending upon short term intake and solar exposure of the mother. Dihydroxylated metabolites of vitamin D constitute an insignificant component of antirachitic properties of milk (Hollis et al.^1981). Since DBP occuring in milk has its origin in the plasma and vitamin D and all the metabolites bind to the protein, DBP entering into milk provides an important route for the transfer of vitamin D and its antirachitic metabolites. Colostrum is extraordinarily rich in plasma proteins (Larson, 1974). Hence colostrum is particularly rich in antirachitic sterols (Oh and Horst, 1981).

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