The Turkish Journal of Pediatrics 35: 205-207, 1993

A CASE OF TRANSIENT HYPERPHOSPHATASAEMIA FOLLOWING VITAMIN D – DEFICIENT RICKETS*

Š. Kutilek MD**, J. Štěpan MD***, M. Bayer MD**

SUMMARY: Kutilek Š, Štěpan J, Bayer M. (Departments of Pediatrics and Internal Medicine Charles University 1st Faculty of Medicine, Prague, Czechoslovakia). A case of Transient hyperphosphatasemia following vitamin-D deficient rickets. Turk J Pediatr 35: 205-207, 1993.

A seven-month-old boy with a diagnosis of vitamin-D deficiency rickets is presented. After treatment with vitamin-D, biochemical and radiological improvement was noted. At 14-months of age the plasma akaline phosphatase activity valve was found to be elevated even though rickets was not present. A diagnosis of hyperphosphatasemia was established. Although rare in infancy, transient hyperphospha tasemia may occur following vitamin-D deficient rickets. This possibility should be considered when evaluating laboratory results in such cases. *Key words: vitamin-D deficient rickets, transient hyperphosphatasemia, alkaline phosphatase.*

Transient hyperphosphatasemia of infancy and early childhood is a rare syndrome characterized by a temporary, isolated elevation of alkaline phosphatase activity (ALP), values in particular the bone isoenzymes. Once coupled with metabolic bone disease, it might concern the physicien, however, such an association is purely coincidental.

Case Report

The patient was a boy of gypsy origin who at birth weighed 2350 g. At the ages of one, three and five months, he was repeatedly treated for bronchopneumonia. Whether the child had received the usual vitamin-D (calciferol) prophylaxis was uncertain, since the family was constantly on the move and also because they were uncooperative.

At the age of seven months, he was admitted to our department because of bronchopneumonia which was treated successfully with Ampicillin. A diagnosis of vitamin D-deficient rickets was established. There was an elevation of bone isoenzyme in serum ALP activity and the X-ray of the wrist showed typical signs of rickets. The boy was treated with four doses of Ergocalciferol (300.000 IU at the age of eight months, dose). He was discharged at the age of eight months, after

* From the Departments of Pediatrics and Internal Medicine, Charles University, 1st Faculty of Medicine, Prague, Czechoslovakia.

** Pediatrician, Charles University, 1st Faculty of Medicine.

*** Internist, Charles University, 1st Faculty of Medicine.

six weeks of hospitalization. The ALP activity had decreased to normal values (Fig. 1) and the wrist X-ray had showed improvement of the rickets.

The boy was followed up for two months. His serum calcium (Ca) and phosphorus (P) concentrations and ALP activity level were within reference ranges. The wrist X-ray taken at the age of nine months showed no signs of rickets. The family did not turn for a periodic follow-up, but the boy was again examined at the age of 14 months.

We were concerned because the serum ALP activity value was 126.9 ukat/1. The serum Ca, P concentrations and ALT, AST, GMT activity values were all within reference ranges and there were no signs of rickets on the wrist X-ray.

The ^{99}Tc bone scan was normal. The boy had no signs of hepatopathy, malabsorption or renal disease. 85% of the increased ALP activity was caused by bone isoenzyme. This activity has rapidly decreased (Fig. 1).

We found no causal relationship between the rickets and hyperphosphatasemia.

Since then, the boy has suffered three episodes of respiratory infections but there were no signs of skeletal or liver involvement.

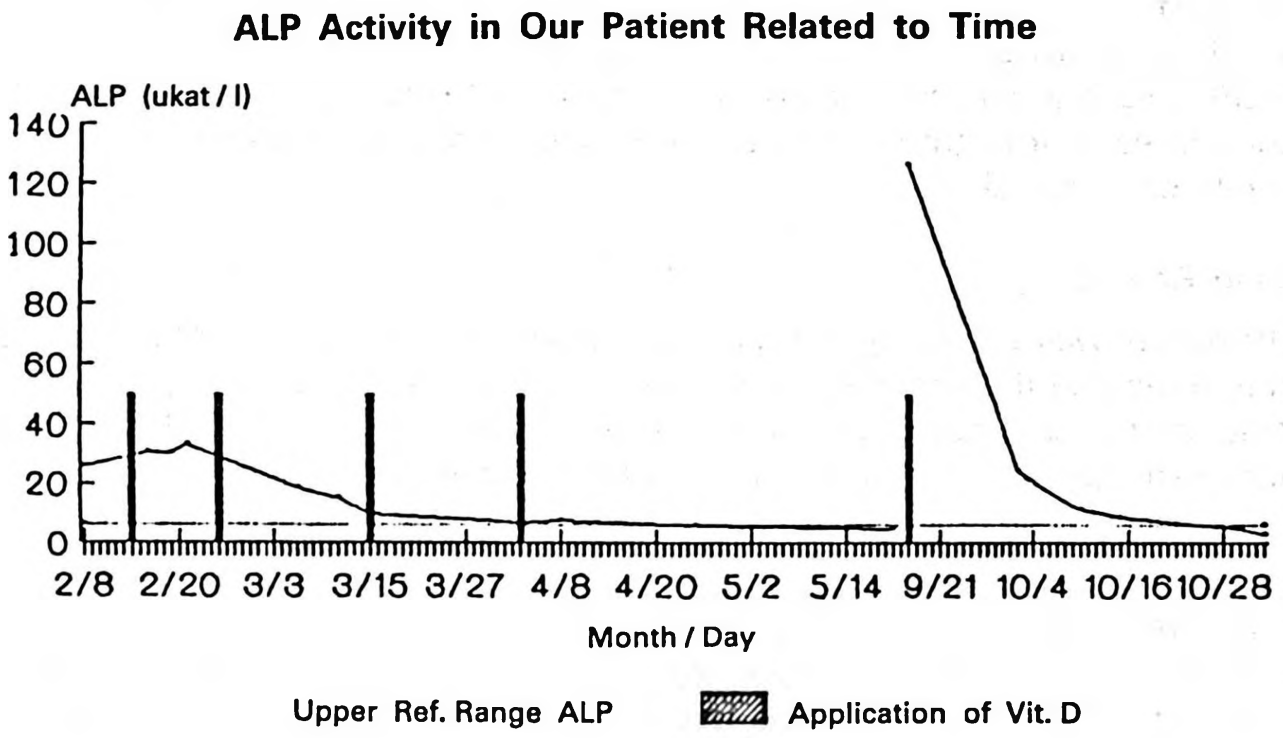

Fig. 1: Change in the serum alkaline phosphatase activity value following vitamin-D treatment and in the follow-up period.

Discussion

The first report of transient hyperphosphatasaemia of infancy and early childhood was described in 1954[1], however more detailed studies and articles have been published in the last decade[2-6].

Kraut et al[4] has devised the following criteria for transient hyperphosphatasemia:

1. An age under five years.
2. Variable symptoms with no relation to ALP increased activity.
3. No evidence of bone or liver disease.
4. Elevation of serum ALP activity in bone and liver.
5. A return to normal of ALP activity valves within four months.

The etiology remains unclear. Three mechanisms may be responsible for the increased ALP activity in serum[6]:

1. Excessive synthesis or release of enzyme from its tissue of origin.
2. Activation of normal amounts of circulating enzyme.
3. Impaired clearance of the enzymes from the circulation.

Frank and Kruse[3] proposed that this syndrome had an infactious origin. A case of transient hyperphosphatasaemia in a girl with vitamin-D dependent rickets was described in 1986[2].

In conclusion, we reported the case of transient hyperphosphatasemia developing after vitamin-D deficient rickets, this with the aim of informing other physicians of this possibility, which is coincidental, but could arise in children with metabolic bone disease.

Abbreviations in The Text

ALP – Alkaline Phosphatase
ALT – Alanine-aminotransferase
AST – Aspartate-amino Transferase
GMT – Gamma-glutamyl transferase

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