

References

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LEON F. KUKLA, CHITRA REDDY, GERALDINE SILKALNS, and MAHENDRA PRASAD

Department of Pediatrics, Martland Hospital, New Jersey Medical School, 65 Bergen Street, Newark, New Jersey 07107, USA.

Correspondence to Dr L. F. Kukla.

Serum 25-hydroxyvitamin D levels in thalassaemia

In patients with hepatic dysfunction low levels of serum 25-hydroxyvitamin D (25-OHD) have been reported (Hepner *et al.*, 1975; Long *et al.*, 1976; Olson *et al.*, 1976; Wagonfeld *et al.*, 1976). These authors stress the importance of further studies in order to correlate the low levels of serum 25-OHD in cirrhotic patients with their bone lesions. It is well known that thalassaemic children have bone lesions and hepatosplenomegaly with evidence of hepatic dysfunction in some cases. This led us to investigate serum 25-OHD in thalassaemia.

Material and methods

Our patients were 36 children, 20 boys and 16 girls, with homozygous β -thalassaemia aged from 5 to 15 years, and 27 controls (19 boys, 8 girls) with the same age range. The controls were chosen from children coming to the hospital for tonsillectomy or

adenoidectomy. In the thalassaemic children blood specimens were taken at least 10 days after the last blood transfusion and haemoglobin was above 8 g/dl.

25-OHD was estimated in the serum of all children by the competitive protein-binding assay as described by Edelstein *et al.* (1973). Venous blood (7 ml) was taken in both groups for routine haematological examination and part of the specimen was used for this study. Serum was separated about half an hour after collection, kept at -20°C , and estimations were performed not later than 7 days after collection.

Blood specimens were collected between the beginning of April and the end of October 1976. We considered as the winter period the first 3 months of observation (1 April-30 June) and the summer period the last 4 months (1 July-31 October).

Results

Table 1 gives the mean values and standard deviations of serum 25-OHD in thalassaemic and control children. As the table shows, thalassaemic children had lower levels of serum 25-OHD than controls ($t=2.4$, $P<0.01$).

Table 2 gives the seasonal variation of serum 25-OHD in both groups. Serum 25-OHD is lower in thalassaemic patients than in the control group at both winter and summer periods. (Winter $t=3.7$, $P<0.0025$; summer $t=1.7$, $P<0.05$.) Serum levels of 25-OHD in both groups were higher during summer. In the thalassaemic group this difference was more marked ($t=2.9$, $P<0.005$).

Table 1 Serum 25-OHD in control and thalassaemic children

| | No. of cases | Serum 25-OHD (ng/ml) (mean \pm SD) |
|-----------------------|--------------|--------------------------------------|
| Controls | 27 | 18.6 \pm 8.6 |
| Thalassaemic children | 36 | 12.8 \pm 9.9 |

Table 2 Seasonal variation of serum 25-OHD in the two groups

| | Serum 25-OHD (ng/ml) (mean \pm SD) | |
|-----------------------|--------------------------------------|----------------------|
| | Winter period | Summer period |
| Controls | 14.0 \pm 5.4 (9) | 20.8 \pm 9.0 (18) |
| Thalassaemic children | 5.6 \pm 4.4 (10) | 15.5 \pm 10.0 (26) |

Number of patients in parentheses.

Discussion

Two factors responsible for the levels of 25-OHD in the serum are the dietary intake of vitamin D and the exposure to sunlight. The first would not account for the difference between controls and thalassaemic children because the dietary intake of vitamin D was the same in both groups. The second accounts only for the differences in both groups between the values in summer and winter and confirms the findings of Stamp (1973) that there are seasonal variations in serum levels of 25-OHD in healthy subjects. A further reason for the low levels of 25-OHD may be an impairment of the hydroxylation of vitamin D in the liver. This was not present in the thalassaemic children, however, because the marked increase of the serum levels during the summer shows that this function of the liver was not severely impaired.

The remaining factor therefore is malabsorption of vitamin D from the gut of thalassaemic children. During winter the main source of vitamin D is food and an impairment of absorption can cause low values of 25-OHD in the serum, while during summer the body does not need this source. It has been shown that in primary biliary cirrhosis there is impaired absorption of vitamin D (Compston and Tompson, 1977; Skinner *et al.*, 1977) and it is likely that a similar mechanism operates in thalassaemia. These findings suggest that the bone lesions found in thalassaemic children may be related in part to vitamin D deficiency and that further investigation is necessary.

Summary

Serum 25-hydroxyvitamin D levels were measured in 36 thalassaemic children and 27 controls aged 5–15 years. Blood specimens were collected from the beginning of April until the end of October 1976. We considered as the winter period the first 3 months and the summer period the last 4 months. We found that (a) thalassaemic children had lower levels of

serum 25-hydroxyvitamin D than controls; (b) there was a seasonal variation of serum 25-hydroxyvitamin D in both groups; and (c) the thalassaemic children had malabsorption of vitamin D. We suggest that the bone lesions in thalassaemic children are related to vitamin D deficiency.

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STELLA TSITOURA, NINETA AMARILIO, P. LAPATSANIS, S. PANTELAKIS and S. DOXIADIS

Institute of Child Health and Paediatric Unit, Aghia Sophia Children's Hospital, Athens 617, Greece.

Correspondence to Professor S. Doxiadis