Studies on calcium excretion in diabetic children

Damme-Lombaerts, Rita Melania Marcel Maria van

SUMMARY

Forty-seven diabetic children were examined systematically for the presence of hypercalciuria, and this was detected in fifteen of the children.

This finding was the origin of an investigation into the calcium excretion of healthy children. In this group of children the calcium excretion in the urine correlated with the urinary sodium excretion, and with the phosphate intake in the diet but not with the calcium intake in the diet.

Compared with the control group, in which four children showed hypercalciuria, the group of diabetic children had an abnormally high incidence of hypercalciuria ($X^2 = 9.90$; $p < 0.01$). The same conclusion was reached when the diabetic children were compared with a group of twenty-one healthy children who received a diet with a calcium content similar to that of the diabetic children.

During the follow-up period of four years four new diabetic patients developed hypercalciuria. In the total group of nineteen hypercalciuric children, the urinary calcium excretion was dependent on the excretion of glucose in six of the children, but was independent of the glucose excretion in thirteen of the children.

Metabolic acidosis could not be shown to be the cause of the hypercalciuria. No correlation was found between the hypercalciuria and the calcium intake in the diet.

The growth in height of the diabetic children with hypercalciuria was not different from that in the children with a normal calcium excretion in the urine. All diabetic children had a decreased cortical thickness of the second metacarpal bone when compared with normal values from the literature, there was however, no difference between the hypercalciuric and the normocalciuric group.

An oral calcium load test provided evidence for a renal rather than an intestinal origin of the hypercalciuria. Oral administration of indomethacin reduced significantly the calcium excretion, without altering the sodium excretion.

We hypothesise that the hypercalciuria found in the diabetic children is due to a renal tubular defect, caused by abnormally elevated levels of prostaglandins or an increased sensitivity to normal levels of prostaglandins.