The Use of Calcium Carbonate and Calcium Phosphate without Vitamin D in the Management of Renal Osteodystrophy

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The management of renal osteodystrophy remains a difficult problem. Many workers in this field have used vitamin D, but this is potentially dangerous because of the possibility of prolonged hypercalcaemia and metastatic calcification. Davidson and Pendras (1967) have reported deaths due to severe myocardial and pulmonary calcinosis in patients with severe renal failure who were given vitamin D and Mallick and Berlyne (1968) have described widespread arterial calcification in similar patients during vitamin D administration.

Instead of vitamin D we have used large quantities of calcium carbonate and calcium phosphate in the management of renal osteodystrophy in our patients on maintenance haemodialysis. The reason we have tried this method is because it has been shown by Clarkson et al (1970) that in chronic renal failure, positive balances of both calcium and phosphate can be obtained in this way.

We have also shown that during the administration of calcium carbonate in this way, there is a fall in parathormone level.

When giving calcium supplements to our patients, calcium carbonate alone has been used if the plasma phosphate is high and both calcium carbonate and calcium phosphate if the plasma phosphate is normal. Calcium phosphate alone has been used for the occasional patients on maintenance haemodialysis who have developed low plasma phosphate levels.

CALCIUM CARBONATE AND CALCIUM PHOSPHATE HAVE BEEN USED IN SEVERAL DIFFERENT WAYS IN OUR MAINTENANCE HAEMODIALYSIS UNIT

1. Hyperparathyroidism

Firstly in a group of nine asymptomatic patients with radiological evidence of hyperparathyroidism. The plasma alkaline phosphatase was raised in eight of these nine patients. Three patients were given calcium carbonate and calcium phosphate, two patients were given calcium phosphate alone
and four patients, calcium carbonate alone.

In four of the nine patients, the calcium supplements produced a fall in alkaline phosphatase to normal with radiological improvement in one. In three of these four patients, the amounts of calcium supplements given had to be reduced after prolonged administration and finally discontinued because of hypercalcaemia. At this stage, subtotal parathyroidectomy was carried out.

In three other patients, the alkaline phosphatase remained elevated but two of these patients were known to be unreliable with diets, fluid intake and other medications. The remaining two patients have been on maintenance haemodialysis for only one month.

Figure 1 shows data from one of these patients (J.M.) with hyperparathyroidism. With the administration of calcium carbonate and calcium phosphate there was a dramatic fall in the alkaline phosphatase level. Eventually however, the calcium supplements were discontinued after approximately one year's treatment because of hypercalcaemia when subtotal parathyroidectomy was carried out.

![Graph showing calcium and phosphorus levels over time.](image)

**Figure 1.** Use of calcium supplements in a patient (J.M.) on maintenance haemodialysis with secondary hyperparathyroidism. PCA (Plasma calcium); Ca x P (Calcium phosphorus product); Alk. Phos. (plasma alkaline phosphatase); Para (subtotal parathyroidectomy).

Figure 2 illustrates the radiological improvement that occurred in this same patient (J.M.) during the administration of calcium supplements and before subtotal parathyroidectomy.
2. After subtotal parathyroidectomy

Four patients who had received calcium supplements for secondary hyperparathyroidism subsequently underwent subtotal parathyroidectomy. Two of these four patients became normocalcaemic post-operatively and have not required calcium supplements. The other two patients initially became hypocalcaemic post-operatively and calcium supplements have raised the plasma calcium to normal. All four patients have shown radiological improvement. It must be stressed that none of these patients were given vitamin D before or after operation.

3. Low plasma inorganic phosphate syndrome after prolonged maintenance haemodialysis

Three patients have developed low plasma phosphate levels with raised levels of alkaline phosphatase after maintenance haemodialysis for a year or more. None of these three patients had radiological evidence of hyperparathyroidism and in all three, the parathormone levels were normal. The calcium
phosphorus product was less than twenty in all three patients and the plasma phosphate levels were less than 2 mg/100 ml. One patient developed a cough fracture of a rib and stress fractures of two metatarsals. In all three patients, the alkaline phosphatase returned to normal after the administration of calcium phosphate and the fractures in the one patient healed normally. Figure 3 shows data from one of these three patients. During the administration of calcium phosphate, the calcium phosphorus product and the alkaline phosphatase both returned to normal.

4. Prevention of metastatic calcification
We have paid particular attention to the calcium x phosphorus product and have regarded a product of greater than 70 as a medical emergency. Our worst case of metastatic calcification is shown in Figure 4. In order to lower the calcium phosphorus product, aluminium hydroxide or calcium carbonate have been employed.

5. Use in Renal Rickets
Finally we have used calcium supplements in a six year old child with advanced renal rickets who was not on maintenance haemodialysis. This patient was treated for one year with calcium supplements and showed substantial radiological and biochemical improvement.
CONCLUSIONS

We conclude from our experience, that calcium carbonate and calcium phosphate without vitamin D can be used successfully in managing several of the disorders of calcium and phosphorus metabolism that occur in patients with chronic renal failure and on maintenance haemodialysis.

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REFERENCES