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Improvement of Anemia and Secondary Hyperparathyroidism with Erythropoietin Treatment in Hemodialysis Patients

Dear Sir,

Anemia is a severe problem in patients with chronic renal insufficiency. While multiple factors can cause anemia in chronic renal failure, decreased erythropoietin (Epo) production is the most important cause [1]. Another cause may be secondary hyperparathyroidism that has not been clearly explained. It was suggested that parathormone (PTH) may play a role by its direct inhibitory effect on erythropoiesis and/or induction of bone marrow fibrosis [2]. We studied the effect of Epo therapy on secondary hyperparathyroidism in hemodialysis (HD) patients.

Twenty-four HD patients who were treated with Epo, 9 males and 15 females, mean age 45.6 ± 14.5 years (range: 18–75) were included in the study (group I). The control group consisted of 17 age- and sex-matched HD patients who were not prescribed Epo (group II). Patients are dialyzed for 4 h, 2–3 times a week. All patients received calcium carbonate (3–9 g/day), calcitriol (0.25 mg/day), and a low-protein diet consisting of 800 mg phosphorus. Group I patients were treated with Epo 60–70 IU/kg s.c. twice a week. In all patients, serum calcium (Ca), phosphorus (P) and alkaline phosphatase (ALP) values were measured monthly, and N-terminal PTH values were measured every 3 months. The serum values of Ca, P, ALP and PTH at baseline and at the third month were compared in the two groups by the Student's t test. All the measurements are shown in table 1.

Table 1. Serum levels of Ca, P, ALP and PTH in groups I and II

	Group I Epo and ($CaCO_3$ + vitamin D)	Group I ($CaCO_3$ + vitamin D)
Ca, mg/dl		
Baseline	8.4 ± 1.0	8.5 ± 1.2
	***	
After treatment	9.2 ± 0.8	8.6 ± 1.3
P, mg/dl		
Baseline	5.9 ± 1.3	7.5 ± 2.2
		**
After treatment	5.1 ± 1.6	6.3 ± 1.7
ALP, IU/l		
Baseline	238.6 ± 134.5	233.2 ± 108.2
	****	
After treatment	172.8 ± 84.7	196.9 ± 104.8
Ca, mg/dl		
Baseline	489.7 ± 452.3	238.0 ± 437.7
	*	
After treatment	327.4 ± 349.3	230.5 ± 295.8

* $p < 0.05$; ** $p < 0.03$; *** $p < 0.01$; **** $p < 0.001$.

With Epo administration in group I, the mean hematocrit value rose from 18.6 ± 2.6 to 24.8 ± 3.5% ($p < 0.001$) while the serum value of Ca increased significantly ($p < 0.001$). The serum values of ALP and PTH decreased significantly ($p < 0.001$, $p < 0.05$). The mean serum value of P also decreased, but the difference was not significant ($p > 0.05$). In group II, the mean serum values of P, ALP and PTH decreased but the difference was not significant with the exception of the serum P values ($p < 0.03$).

A number of studies have shown that one of the major causes of renal osteodystrophy is excessive PTH secretion in chronic renal failure and that this status may contribute to the anemia seen in uremic patients [3]. Improved anemia in patients with primary hyperparathyroidism has been reported after surgical ablation of parathyroid adenoma [4]. Also, it has been noted that parathyroidectomy may improve the anemia in patients with chronic renal failure. Unfortunately, observations on PTH-related anemia

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are poorly understood. Excess secretion of PTH induces bone marrow fibrosis, and this process could reduce erythropoiesis or inhibit the renal and/or extrarenal production of Epo [5, 6].

As we found a significant improvement in the serum levels of Ca, ALP and PTH in patients treated with Epo, our data confirm that Epo treatment may improve secondary hyperparathyroidism with correction of anemic in HD patients. Moreover, we think that there may be a negative relationship between PTH and Epo, and the same hypothesis could also be possible between Ca and Epo.

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