Decreased Blood Pressure after a Year of Erythropoietin

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Dear Sir,

The most frequent secondary effect of erythropoietin (EPO) treatment is exacerbation or the appearance of arterial hypertension (AH), in some cases with important complications [1,2]. There are few studies on the development of blood pressure after the correction of anemia with EPO, and an increment in antihypertensive drug requirements is the most extended observation at short follow-up [3–5].

We studied 14 patients (8 males), aged 40 ± 14 years, hemodialysis (HD) time 80 ± 48 months, who were started on EPO (50 IU/kg i.v. after HD) adjusting for a hematocrit of 30–35%. Systolic arterial pressure (SAP), diastolic arterial pressure (DAP), and mean arterial pressure [MAP = $DAP + 1/3 (SAP - DAP)$] were calculated as mean $\pm$ SD of daily pre-HD determinations on a monthly basis.

Before EPO, 10 patients required drugs to maintain SAP below 160 mm Hg and/or DAP below 95 mm Hg. After 3 months of EPO, 5 patients required a higher antihypertensive dosage, and another 1 needed hypotensives. 5 patients continued with the same treatment, and the other 3 remained normotensive. From months 3 to 12, SAP and MAP decreased slowly, being significantly lower at 12 months, (table 1). DAP also diminished, but not significantly. At the end of the study, 7 patients were normotensive without drugs (3 from the beginning and 4 initially treated). Comparing these 7 patients with the other 7 requiring drugs, there were no significant differences (χ^2 test) in age, sex, time on HD, dry weight,

Table 1. Evolution of hematocrit (Ht) SAP, DAP, and MAP in hemodialyzed patients on EPO

hematocrit (31.6 ± 6.5 vs. $32.4 \pm 3.8\%$, NS) or EPO dose (48 ± 11 vs 60 ± 22 IU/kg/HD, NS).

The causes of AH in EPO treated patients have not been completely elucidated, but hemodynamic changes induced by the correction of anemia are probably incriminated. This correction is accompanied by increased vascular peripheral resistances and blood viscosity, with subsequent increment in blood pressure [2]. Half of our patients with AH before EPO required higher doses of hypotensives at 3 months. Curiously, after 10 months of stabilization of hematocrit levels, SAP and MAP had decreased to values significantly lower than basal ones, allowing the withdrawal of the drugs in some patients. Probably, the hemodynamic stabilization after the correction of anemia produced a lowering in peripheral resistances, maintaining adequate cardiac output and concomitant decreases in SAP and MAP. It seems likely that the

very slow correction of anemia with EPO allows blood pressure stabilization, or even a decrease at midterm, with obvious benefits for the patients.

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References

Mayer G, Stefenelli T, Cadar EM: Blood pressure and erythropoietin. *Lancet* 1988;i:351–352.

Rayne AEG: Hypertension, blood viscosity, and cardiovascular morbidity in renal failure:

Implications of erythropoietin therapy. *Lancet* 1988 ;i:97–99.

Eschbach JW, Downing MR, Egrie JC, Browne JK, Adamson JW: USA multicenter clinical trial with recombinant human erythropoietin: Results in hemodialysis patients. *Contrib Nephrol* 1989;76:160–165.

Suzuki M, Hirasawa Y, Hirashima K, et al: Dose-finding, double-blind clinical trial of recombinant human erythropoietin

in Japanese patients with end-stage renal disease. *Contrib Nephrol* 1989;76:179–198. 5 Teruel JL

Pascual J

Jimenez M

et al: Hemodynamic changes in hemodialyzed patients during treatment with recombinant human erythropoietin. *Nephron*

in press.