Enalapril-Associated Anemia in a Patient with IgA Nephropathy and Hypertension

J.W. van der Pijl
R.T. Krediet
Renal Unit, Department of Medicine, University of Amsterdam, The Netherlands

J.W. van der Pijl, MD, Renal Unit, Department of Medicine, Academic Medical Centre, Room F4-215, University of Amsterdam, Meibergdreef 9, NL-1105 AZ Amsterdam (The Netherlands)

Fig. 1. Time course of hemoglobin and creatinine in relation to antihypertensive treatment in the male, 45-year-old patient.

Although anemia is often not mentioned as a possible side-effect of CEI [6], we conclude that the existence of enalapril-associated anemia must be considered when this complication develops during enalapril treatment.

References
Griffing GT, Melby JC: Enalapril (MK-421) and the white cell count and hematocrit. Lancet 1982;i:1361.

Dear Sir,
Enalapril has been associated with minor decreases in hematocrit in a small group of volunteers [1], and a few reports have focused attention on a reduction in hematocrit in renal transplant recipients [2,3] and also in patients on chronic hemodialysis [4].

We report enalapril-associated anemia in a patient known to have IgA nephropathy. A 45-year-old white male attended the outpatient clinic in August 1990 because of severe hypertension. Laboratory investigations showed a hemoglobin level of 13.4 g/dl and a plasma creatinine of 179 µmol/l. Enalapril, furosemide and nifedipine were prescribed and later on adjusted according to blood pressure and complaints of orthostatic hypotension. A normochromic, normocytic anemia was found in November 1990 (fig. 1). No explanation of this anemia was found: WBC and platelets were within the low normal range, reticulocytes 2%, haptoglobin 0.3 g/l, ferritin, folate acid, vitamin B12, lactate dehydrogenase and bilirubin were also normal. Antinuclear antibodies were positive, without a positive LE cell phenomenon nor positive antibodies to dsDNA. A bone marrow aspiration and biopsy showed a normocellular aspect. After discontinuation of enalapril, hemoglobin rose to normal values. A rechallenge with a lower dose, given after 3 months, provoked anemia again.

Animal studies [5] have pointed out that the ischemic response elicited by angiotensin II might be an important stimulus for the production of erythropoietin, which could be overruled by giving an angiotensin-converting enzyme inhibitor (CEI). The observations [4] done in chronic hemodialysis patients offer also support for this finding, because strong
correlations were found between angiotensin II, erythropoietin and the reticulo-cyte count before and during therapy with CEI.

