Bone Marrow Involvement in Systemic Amyloidosis: Another Mechanism for Erythropoietin Resistance

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Dear Sir,

The introduction of recombinant human erythropoietin (r-HuEPO) in clinical practice is a major advance in the treatment of renal anemia [1]. With increasing experience, several mechanisms for resistance to r-HuEPO have been described [2]. Systemic amyloidosis is associated with diverse hematologic manifestations but to our knowledge, it has not been held responsible for r-HuEPO resistance [3]. We are reporting a chronic hemodialysis patient with systemic amyloidosis due to familial Mediterranean fever (FMF) who exhibited resistance to r-HuEPO treatment.

A 40-year-old man who had received a diagnosis of FMF developed nephrotic syndrome in 1985. Renal biopsy revealed renal amyloidosis in 1986, and he was admitted to our hemodialysis unit in 1990. He was dialyzed 3 times weekly and received r-HuEPO, iron and folic acid supplementations as a part of his treatment. The initial dose of r-HuEPO was 4,000 U 3 times weekly and was increased to 6,000 U 3 times weekly because of nonresponse. His hemoglobin (Hb) and hematocrit (Hct) values remained below 85 g/dl and 24%, respectively, after 16 months of treatment. The route of administration was intravenous injections in the beginning and was switched to subcutaneous injections, but this did not affect the resistance to r-HuEPO. Physical examination was normal. A detailed hematological survey revealed the following

findings: WBC: $6.8 \times 10^9/l$; platelets: $188 \times 10^9/l$; MCV: 92 μ^3 , serum ferritin: 167 $\mu g/l$; serum iron: 13.94 $\mu mol/l$; serum-iron binding capacity: 49.31 $\mu mol/l$; red cell folic acid: 2,016 nmol/l cells. Macrophage iron stores were normal and the bone marrow was normocellular in the bone marrow aspiration examinations.

There was no clinical or laboratory evidence for uncontrolled renal osteodystrophy.

The bone marrow biopsy when stained for amyloidosis showed diffuse infiltration with amyloid (fig. 1,2) and it was concluded that it was responsible for the r-HuEPO resistance in this patient.

References

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