## Bone marrow amyloidosis

**Haematologica 2006;** 91:(1)e16

A 54-year-old man presented with nephrotic syndrome, renal impairment and restrictive cardiomyopathy. On admission, physical examination revealed pallor, peripheral edema and moderate hepatosplenomegaly. Hematologic values were: Hb 9.7 g/dL, WBC $10.4\times10^9$/L with a normal differential count, platelets $109\times10^9$/L. Kappa monoclonal light chains were identified by urine immunofixation. Circulating kappa free light chains were 378 mg/L and lambda 56.3 mg/L. Bone marrow aspirate displayed slightly hypocellular marrow with maturing hematopoietic progenitors and mild dyserythropoiesis. There were 8% morphologically normal plasma cells. Various-sized clumps of pink amorphous material were scattered on the smears (Figure, A and B). These deposits stained with Congo red (Figure, C), that under polarized light produced a characteristic apple-green birefringence (Figure, D). Abdominal fat pad aspirate confirmed the presence of amyloid. Therefore, a diagnosis of AL amyloidosis with renal, cardiac and bone marrow involvement was made. The patient was treated with high-dose dexamethasone with progressive improvement of his condition.

Systemic AL amyloidosis is a plasma cell dyscrasia in which the fibril amyloid protein is produced by monoclonal plasma cells and consists of whole or fragments of immunoglobulin light chains.[1] It is associated with plasma cell myeloma in about 15% of cases. In the other cases a moderate monoclonal increase in plasma cells is usually present in the bone marrow.[2] A monoclonal immunoglobulin is found in the serum or urine in more than 80% of patients. Amyloid deposits are detected in blood vessels and as interstitial foci in bone marrow sections in approximately 60% of patients,[2,3] but very rarely, and only when there is extensive bone marrow involvement, extracellular amyloid clumps are present in a bone marrow aspirate.

*Rosangela Invernizzi,[1] Giovanni Palladini,[1,2] Chiara Benatti,[1] Erica Travaglino,[1] Mario Nuvolone,[2] Giampaolo Merlini[2]*

*[1]Department of Internal Medicine, [2]Center for Amyloidosis, Biotechnology Research Laboratories, University of Pavia and IRCCS Policlinico S. Matteo, Pavia, Italy*

*Correspondence: R. Invernizzi, M.D.,*
*Clinica Medica III, Policlinico S. Matteo, Piazzale Golgi, 27100 Pavia (Italy)*
*E-mail: r.invernizzi@smatteo.pv.it*

### References

1. Merlini G, Bellotti V. Molecular mechanisms of amyloidosis. N Engl J Med 2003; 349:583-96.
2. Kyle RA, Gertz MA. Primary systemic amyloidosis: clinical and laboratory features in 474 cases. Semin Hematol 1995; 32:45-59.
3. Swan N, Skimmer M, O'Hara CJ. Bone marrow core biopsy specimens in AL (primary) amyloidosis. Am J Clin Pathol 2003; 120:610-6.