



## Letters to the Editor

### Hematologists and nephrologists working together: moving forward with a new integrated care model for blood-related malignancies?

**TO THE EDITOR:** Patients with hematological malignancies (HM) are at high risk of renal complications [1, 2]. Additionally, HM may occur in people with a pre-existing renal impairment (RI). Indeed, in patients with HM, several forms of RI may arise from the underlying disease, the adverse effects of antineoplastic therapies [1], or late clinical complications [2]. On the other hand, some forms of RI may be detectable during the initial diagnostic work-up for HM as the result of comorbid illnesses, such as hypertension or diabetes, which are typically observed in older individuals [3]. In addition, most HM tend to occur in elderly, in parallel with the age-related decline in renal function. The presence of a RI at the onset of disease has been recognized as an independent prognostic factor in the case of newly diagnosed diffuse large B-cell lymphoma [4] and multiple myeloma (MM) [5]. Furthermore, in patients with acute myeloid leukemia (AML), RI can be an insurmountable barrier to administering an appropriate and effective chemotherapy for optimal management [6]. Given the high incidence of RI, the baseline renal function should be accurately assessed in the initial work-up of a newly diagnosed HM, including even the patients with normal serum creatinine (sCr) levels. This assessment allows physicians to make appropriate choices for treatment such as adjusting the dosage of the chemotherapeutic agents [1, 3], novel antineoplastic targeted compounds, antibiotics, or analgesics [7].

The coexistence of HM and renal disorders interrupt the optimal antineoplastic treatments because the pharmacological behavior of administered drugs and their active compounds may be influenced by the renal function. In order to unintended toxic effects due to an altered metabolism or a compromised renal excretion, it is important that physician fully understand the characteristics of drugs. Although

our knowledge on this issue has improved in recent years, the management of patients with HM accompanying RI is challenging due to the lack of organizational structures and collaborative models between nephrologists and hematologists. Additionally, patients with RI are commonly excluded from preclinical development or phase I trials [8] because they are considered to be at high risk of complications. Therefore, a comprehensive team approach such as 'hematonephrology' or 'nephrohematology' is required to appropriately manage these vulnerable patients [9].

Would now be the time to think about reorganizing the hospital wards and providing integrated services for patients with HM by clinical teams composed of hematologists and nephrologists? In our opinion, this question should be addressed by operational trials involving nephrologists and hematologists working in the same team developed for patients with HM. In this comprehensive team, patients with HM could receive more specialized and constant nephrologic management throughout the course of the disease. Prospective studies could demonstrate the evidence for the clinical effectiveness and cost-effectiveness of early referral strategies for the management of these patients with or without evident markers of renal disease. This would optimize the treatment and prevent the progression of RI to more advanced stages through the use of chemotherapeutic agents and/or other antineoplastic agents, which may potentially induce further kidney damage and aggravate an already compromised renal function.

In conclusion, we recommend the development of new departments and clinics where hematologists and nephrologists could manage HM patients together. Thus, we advocate the development of a new and modern medical specialization such as hematonephrology, to improve our knowledge and outcomes of patients with HM and RI.

**Pasquale Niscola<sup>1</sup>, Tommaso Caravita<sup>1</sup>,  
Paola Tatangelo<sup>2</sup>, Agostina Siniscalchi<sup>1</sup>,  
Paolo de Fabritiis<sup>1</sup>, Roberto Palumbo<sup>2</sup>**

<sup>1</sup>Hematology Unit and <sup>2</sup>Nephrology Unit,  
Department of Medical Specialties S. Eugenio Hospital  
(ASL Roma 2), Rome, Italy



**Correspondence to: Pasquale Niscola**  
*Hematology Unit, S. Eugenio Hospital,*  
*Rome 00144, Italy*  
*E-mail: pniscola@gmail.com*

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## A case of synchronous multiple myeloma and chronic myeloid leukemia

**TO THE EDITOR:** Multiple myeloma (MM) is a hematologic malignancy caused by the proliferation of clonal plasma cells in the bone marrow, leading to uncontrolled production of monoclonal immunoglobulin. Chronic myeloid leukemia (CML) is a myeloproliferative neoplasm associated with the presence of the *BCR-ABL1* fusion gene. These are uncommon malignancies that account for approximately 1.6% and 0.4% of all newly diagnosed cancers in the United States, respectively [1]. In Korea, these are also rare disease and the crude incidence rates of those are 2.5/100,000 and 0.8/100,000, respectively [2, 3]. The concurrent diagnosis of MM and CML in one patient is an extremely rare event. In this report, we describe an additional case of synchronous MM and CML.

A 64-year-old man was referred to our hospital for evaluation of a right pleural soft-tissue mass and a sternal osteolytic lesion incidentally detected on chest computed tomography (CT). The patient had no specific symptoms and remarkable findings on physical examinations. A complete blood count showed a white blood cell counts of  $13.1 \times 10^9/L$ , with a differential count of 65% neutrophils, 15% lymphocytes, 10% monocytes, 5.2% eosinophils, and 4% basophils; hemoglobin levels of 8.3 g/dL; and platelet counts of  $234 \times 10^9/L$ . Blood chemistry showed reversal of the albumin-globulin ratio (total protein 11.8 g/dL, and albumin 2.3 g/dL), renal dysfunction (creatinine clearance 38.4 mL/min), and hypercalcemia (calcium 11.8 mg/dL). A total of 7 g/dL of monoclonal gammopathy (IgA lambda type) was detected with serum immunofixation electrophoresis. The ratio of serum kappa/lambda free light chains was 0.006 and serum  $\beta 2$  microglobulin level was 19.8 mg/L. Radiological investigation revealed multiple osteolytic lesions in the skull and axial skeleton. The needle biopsy of pleural soft-tissue mass showed infiltrations of monotonous round cells with eccentrically located nuclei and different sizes, but no evidence of myeloid cell infiltration. Most of the infiltrated cells were positive for CD138 on immunohistochemical staining (Fig. 1). Bone marrow biopsy revealed increased cellularity (up to 90%), consisting of plasmacytoid round cell infiltrations and myeloid cells with varying maturation. A patchy positive reaction for CD138 was compatible with myeloma involvement, and myeloid cells were positive for myeloperoxidase. The bone marrow aspirate demonstrated myeloid hyperplasia with increased eosinophils, basophils and plasma cells. Plasma cells showed mature forms with condensed nuclear chromatin, indistinct nucleoli, and abundant basophilic cytoplasm with a perinuclear halo (Fig. 2). Chromosome analysis revealed 46,XY, t(2:3)(p15;q26), t(9:22)(q34;q11) in 20 of 21 cells, and fluorescent in situ