

Complete haematological response with granulocyte colony stimulating factor (G-CSF) and hydroxyurea in relapsed acute myeloid leukaemia (AML) of the elderly

Lionello Camba, Massimo Bernardi, Alessandra Pescarollo

Ematologia e Trapianto di Midollo Osseo, Ospedale San Raffaele, Milan, Italy.

Correspondence: Lionello Camba, Ematologia e Trapianto di Midollo Osseo, Ospedale S Raffaele, via Olgettina 60, 20132 Milan. Italy. E-mail: lionello.camba@hsr.it



Text

Sastre et al.¹ report on a case of complete response obtained with granulocyte-macrophage colony stimulating factor (GM-CSF) and low dose cytarabine in an elderly patient with refractory AML. We would like to describe an elderly patient with relapsed AML, who responded to G-CSF and hydroxyurea. The patient presented in August 1998, aged 75, with anaemia, leucocytosis and 10% blast cells in the peripheral blood. Bone marrow aspirate confirmed the diagnosis of LMA-M4 and normal cytogenetics. He was treated with the Manchester Protocol (cytarabine 100 mg/m²x2 for 5 days, mitoxantrone 4 mg/m² for 4 days)² and went in to C.R. after the 2nd induction course. He was then consolidated with intermediate dose of Cytosine-arabioside (1 g/m²/day for 6 days) and Idarubicin (10 mg/m²/day for 3 days). He relapsed after 16 months in January 2001. Because of his age he was treated conservatively with blood and platelet transfusions and put on Hydroxyurea to contain his leukaemia. G-CSF was started for his severe neutropenia. The haematological indices improved gradually, he became transfusion-independent, and by week 12 from starting treatment he was in C.R. without haematological or other complications (Table). G-CSF was then reduced. He is currently on 300 mg 10 days and HU 0.5 g/day.

Achievement of C.R. in elderly patients with G-CSF with and without chemotherapy has been described.^{2,3} G-CSF is used in elderly AML patients to achieve rapid neutrophil recovery and to prime myeloblast growth to the cytotoxic action of chemotherapy⁴ but C.R. rate and long term survival are not convincingly affected.^{5,6} Large studies on the use of G-CSF or GM-CSF with non-myelotoxic low dose chemotherapy in elderly AML patients have not been systematically undertaken. Such studies might be able to establish whether this approach is worth pursuing: the high degree of compliance, the absence of myelotoxicity, the outpatient-only management make this treatment ideal for such patient population. Finally, why in a number of patients G-CSF is able to induce C.R. by restoring an effective haemopoiesis and repressing the leukaemic proliferation remains unknown. Differentiation of blast cells and stimulation of the non-leukaemic cell population may be hypothesised.⁷



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