onset of venous thrombosis during oral contraception. Clin Appl Thromb Hemost 1995; 1:118-9.
8. Girolami A, Simioni P, Girolami B, Radossi P. Homozygous patients with APC resistance may remain paucisymptomatic or asymptomatic during oral contraception. Blood Coagul Fibrin 1996; 7:590-4.
9. Simioni P, Prandoni P, Girolami A. Low rate of venous thromboembolism in asymptomatic relatives of probands with factor V Leiden mutation. Ann Intern Med 1999; 130:538.
10. Girolami A, Simioni P, Sartori MT, Zanardi S. Oral contraceptives caused thrombosis in a monoovular twin with protein C deficiency, while the other, without medication, remained asymptomatic. Blood Coagul Fibrinol 1992; 3:119-20.
11. Girolami A, Stevanato F, Lazzaro AR. Bilateral ileofemoral thrombophlebitis after ten contraceptive pills in a 25-year-old woman with antithrombin III deficiency. Acta Haematol 1988; 79:118-9.
12. Esmon CT, Gu JM, Xu J, Qu D, Stearns-Kurosawa DJ, Kurosawa S. Regulation and functions of the protein C anticoagulant pathway. Haematologica 1999; 84:363-8.

Refining prognosis of acute myeloid leukemia patients

Estey *et al.*[1] present interesting data suggesting that the prognosis within each of the cytogenetic subsets of acute myeloid leukemia (AML) needs to be refined. Mandelli *et al.*,[2] in this journal, recently discussed the role of genetic characterization in the therapy of AML, and the investigative efforts needed for the design of tailored treatment for each and every AML patient. They concluded that the prognostic role of genetic lesions, currently identified by karyotyping studies, needs to be validated in large series of AML patients prospectively characterized by advanced molecular/cytogenetic analyses and treated uniformly. In addition, searches for new clinically relevant genetic abnormalities, and diagnostic tools for their rapid identification are urgently needed to identify prognostic categories better. Other studies in this journal have emphasized the same need in AML and myelodysplastic syndromes.[3-8] The final target is, however, to identify the AML gene alterations in order to develop new drugs targeted to the specific lesion in the individual patient.

References

1. Estey EH, Pierce S, Keating MJ. Identification of a group of AML/MDS patients with a relatively favorable prognosis who have chromosome 5 and/or 7 abnormalities. Haematologica 2000; 85:246-9.
2. Mandelli F, Petti MC, Lo Coco F. Therapy of acute myeloid leukemia: towards a patient-oriented, risk-adapted approach. Haematologica 1998; 83:1015-23.
3. Bassan R, Raimondi R, Lerede T, et al. Outcome assessment of age group-specific (+/- 50 years) post-remission consolidation with high-dose cytarabine or bone marrow autograft for adult acute myelogenous leukemia. Haematologica 1998; 83:627-35.
4. Sanz GF, Sanz MA, Greenberg PL. Prognostic factors and scoring systems in myelodysplastic syndromes. Haematologica 1998; 83:358-68.
5. Balduini CL, Guarnone R, Pecci A, Centenara E, Invernizzi R, Ascri E. The myelodysplastic syndromes: predictive value of eight prognostic systems in 143 cases from a single institution. Haematologica 1999; 84: 12-6.
6. Estey EH. Prognosis and therapy of secondary myelodysplastic syndromes. Haematologica 1998; 83: 543-9.
7. Cazzola M, Anderson JE, Ganser A, Hellström-Lindberg E. A patient-oriented approach to treatment of myelodysplastic syndromes. Haematologica 1998; 83:910-35.
8. Ferrara F, Annunziata M, Copia C, Magrin S, Mele G, Mirto S. Therapeutic options and treatment results for patients over 75 years of age with acute myeloid leukemia. Haematologica 1998; 83:126-31.