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The unique clinical features and distinctive hemostatic dysfunction in acute promyelocytic leukemia

A cute promyelocytic leukemia (APL) has distinct molecular, morphologic and clinical characteristics that set it apart from other forms of acute myelogenous leukemia. The majority of patients at presentation have varying degrees of hemostatic and fibrinolytic abnormalities, resulting in increased thrombotic and bleeding complications. The coagulation profile shows signs of activated coagulation with increased fibrinopeptide A, prothrombin fragment 1+2 and thrombin-antithrombin complexes and of consumption of coagulation factors with increase fibrin D-dimer and decreased fibrinogen. Fibrinolytic abnormalities are also present with increased t-PA, u-PA, and plasmin and decreased PAI-1 and a2-antiplasmin. The excessive fibrinolysis is characterized by increased expression of u-PA, t-PA and the annexin A2/S100A receptor in the leukemic promyelocytes. Thrombin activatable fibrinolytic inhibitor (TAFI) is reduced. These changes are reversed by differentiation therapy with ATRA or arsenic trioxide. The clinical bleeding from coagulopathy generally abates by 5-7 days, but the abnormal coagulation and fibrinolytic profiles return to normal after 14 days or longer. Though differentiation therapy results in an improved clinical outcome and a molecular remission in over 90% of patients, the early death rate in the first 30 days of diagnosis remains unacceptably high at 5-10%. The major cause of this is bleeding, commonly as intracranial hemorrhage. The latter may be related to increased expression of annexin A2 in the cerebral microvasculature. Unfortunately, an effective therapeutic approach to correct the bleeding complications is still elusive. The APL patient has also an increased thrombors risk of up to 10%, an incidence that has not been changed by differentiation therapy.

Biography

Hau C Kwaan completed his MBBS in 1952 and MD in 1958 from University of Hong Kong. He completed his fellowship in Columbia University College of Physicians and Surgeons in New York in 1959. He is board certified in Internal Medicine, Hematology and Medical Oncology. He is the Marjorie C Barnett Professor of Hematology-Oncology and Professor of Medicine at the Feinberg School of Medicine, Northwestern University. He has published more than 400 papers in peer reviewed journals and edited 3 books on Thrombosis. He is the Senior Editor of the seminars in Thrombosis and Hemostasis.

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