

Addendum

The Unique Hemostatic Dysfunction in Acute Promyelocytic Leukemia

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Addendum

The author would like to include the following additional information, as pertaining to the above-mentioned article. The omission of this data from the main article is regretted.

Note

An important recent finding into the pathogenesis of increased fibrinolytic bleeding in acute promyelocytic leukemia is the role of S100A10 protein (also known as P11). P11 is found to co-localize with plasminogen-Rkt and urokinase receptor¹ and is part of the mechanism for activation of fibrinolysis. It is also found on the surface of acute promyelocytic leukemia cells,² binds tissue plasminogen activator and plasminogen, and is downregulated with all-trans-retinoic acid treatment.

References

- 1 Miles LA, Parmer RJ. Plasminogen receptors: the first quarter century. *Semin Thromb Hemost* 2013;39(4):329–337
- 2 O'Connell PA, Madureira PA, Berman JN, Liwski RS, Waisman DM. Regulation of S100A10 by the PML-RAR- α oncoprotein. *Blood* 2011;117(15):4095–4105