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Hemostatic complications of angiogenesis inhibitors in cancer patients

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Abstract

Tumor vasculature and tumor-associated neo-angiogenesis have recently become major targets for rational drug design of antineoplastic agents. Five such agents with angiogenesis inhibiting activity (thalidomide, lenalidomide, bevacizumab, sunitinib, sorafenib) have already obtained US Food and Drug Administration approval for clinical use and many others have entered clinical trials. Vascular complications, including venous or arterial thromboembolism and hemorrhage, have emerged as relevant toxicities in several clinical trials with angiogenesis inhibitors. Given the well-known interplay between the blood clotting system, angiogenesis, and tumor growth, a better understanding of the impact of these new drugs on overall hemostatic balance is required. In this brief overview, we discuss the incidence of hemostatic complications, the likely pathogenetic mechanisms involved, and the critical need to establish in randomized clinical trials the usefulness of thrombosis prophylaxis to prevent these complications. Careful documentation of hemostatic complications during treatment with each of the new antiangiogenic drugs is warranted. Further studies are urgently required to better define the causal association of these new agents with hemostatic complications and to establish the best prophylactic strategy.