ABSTRACT FORM ECAT SYMPOSIUM 8 – 9 NOVEMBER 2018

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Title:

Crossroads between cancer and coagulation

Abstract:

In cancer patients, the risk of venous thrombosis is strongly increased. The incidence of cancer-associated thrombosis (CAT) has been steadily rising over the past two decades. CAT aggravates the clinical course of cancer patients, reduces quality of life, can interrupt cancer treatment and has a profound impact on mortality. Prophylactic anticoagulation in these patients is not feasible because of risks of (fatal) bleeding. As the etiology of CAT remains largely unknown, treatment of CAT, as well as prediction of which cancer patients may develop CAT and are eligible for anticoagulation are severely hampered. We have recently delivered proof of principle that tumor-expressed genes associate with an enhanced risk of CAT and that these genes may be used to identify cancer patients that are at risk of CAT. Finally, some of these genes may form excellent targets to prevent CAT in cancer patients without introducing a bleeding risk.

Conversely, blood clotting also appears to promote metastasis through a yet unknown mechanism. We recently obtained insightful results that indicate that blood clotting factors influence integrin function and early prometastatic events such as cancer stem cell formation, both in patient material and in preclinical models. In addition, blood clotting factors appear to influence prometastatic programs such as epithelial-to-mesenchymal transition (EMT). Inhibition of these clotting factors with monoclonal antibodies reduces metastasis and prometastatic features of the tumor.

In conclusion, cancer and coagulation are involved in a vicious cycle that can be targeted to prevent CAT and to stem cancer progression.