Anticoagulants Inhibit Breast Cancer Cells from Exploiting Platelets

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Public Abstract:

The role of platelets in controlling bleeding is well known. Recently however, the potential responsibilities of the platelet has increased as more has been learned about their abilities to multitask. One such role is in establishing cancer metastasis. It has been shown that platelets and tumor cells “communicate” with each other aiding in tumor growth and metastasis formation. Platelets have been shown to be essential in helping tumor cells traverse through the blood stream to set up new sites for metastasis formation. The link between platelets and malignancy has been appreciated for years. Patients with cancer have increased risk for developing blood clots termed thrombosis, this risk is especially high in breast cancer patients. These patients are treated with heparin, a anticoagulation medication aimed at blocking clotting pathways. Clinical studies evaluating the role of these medications revealed that those patients who received anticoagulation medication not only had a decreased risk of developing a repeat clotting event but also had a prolonged survival and less metastatic disease formation. Recently, work in our laboratory has implicated platelets in aiding the new blood vessel formation (angiogenesis) that is essential for feeding tumor growth and metastasis formation. When platelets come into contact with breast cancer cells, the platelet releases factors into the blood that promote angiogenesis. We have found that release of these factors can be blocked by treating the platelets with anticoagulation medications prior to exposing them to breast cancer cells. In this proposal we aim to better understand how anticoagulation medications disrupt the interplay between breast cancer cells and platelets. Our goal is to explore the mechanisms by which anticoagulants disrupt the communication between platelets and breast cancer cells. We will use this knowledge to explore the platelet’s role in patients with breast cancer and how this is impacted by anticoagulant medication. Ultimately, this work will lead to the use of targeted platelet therapies that can hijack the platelets’ role as a mediator of angiogenesis leading to decreased tumor growth and inhibition of metastatic disease formation in breast cancer. As more is learned about the interaction between platelets and tumor cells, new avenues for interfering with their relationship will emerge. These studies would set the stage for the development of a new class of drugs that can aid in our battle against metastasis and help to extend the lives of breast cancer patients.