An ultrastructural and viscoelastic study of the hypercoagulable state of breast cancer patients and the implications for the disease

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Abstract (300-word limit)

Statement of the Problem: Breast cancer patients are at an increased risk for thrombotic events such as deep vein thrombosis (DVT) and venous thromboembolism (VTE), drastically affecting survival and quality of life post-treatment for these patients. It has been proposed that this increased risk is caused by cancer associated inflammation-induced hypercoagulation, a key factor involved in thrombus formation. Methodology & Theoretical Orientation: This study utilized microscopy and rheological techniques to examine coagulation components during clot formation, in order to obtain a better understanding of how changes to these components may increase thrombus formation and thus the risk of thrombotic events. Whole blood from treatment-naïve breast cancer patients were compared to whole blood from healthy controls. Routine clinical tests were used to obtain an overall clinical picture of each participant. Scanning electron microscopy was used to study the fine ultrastructure of the red blood cells and platelets. Thromboelastography (TEG) was used to study the changes in clot dynamics during coagulation. Findings: SEM showed platelets to be activated as well as a presence of spontaneous fibrin fibre formation. Also, red blood cells from the patient group showed more irregular surface membranes, increased agglutination and eryptosis when compared to healthy controls. Results from the TEG showed that clots form faster in breast cancer patients, with increased strength and rigidity, thus revealing the hypercoagulable nature of whole blood in this patient group. The results in this study have revealed the marked differences in coagulation and associated blood components between healthy controls and treatment-naïve breast cancer patients. Conclusion & Significance: They provide a greater understanding of clot formation dynamics and has shown that even in a small sample size, breast cancer patients are at an increased risk of thrombotic events, traceable through rheological techniques. This justifies further investigation into the utilization of these techniques in a clinical, point-of-care setting, in order to increase the chance of survival and quality of life for these patient's post-treatment.

Image

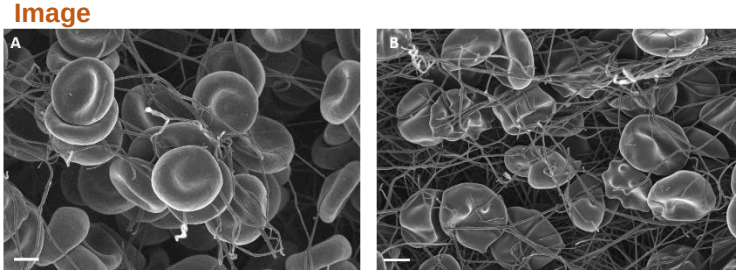


Image 1: Scanning electron images of whole blood. A: Healthy control group images showing normal red blood cells B: Breast cancer group images showing abnormal deformed red blood cells. Scale: 1µm

Recent Publications

1. DeNardo, D. G. and L. M. Coussens (2007). "Inflammation and breast cancer. Balancing immune response: crosstalk between adaptive and innate immune cells during breast cancer progression." Breast Cancer Research 9(4): 1.
2. Lima, L. G. and R. Q. Monteiro (2013). "Activation of blood coagulation in cancer: implications for tumour progression." Bioscience reports 33(5): e00064.
3. Khorana LiX, SchwachaMG, ChaudryIH, ChoudhryMA (2008). Acutealcohol intoxication potentiates neutrophil-mediated intestinal tissue damage after burn injury. Shock 29:377.
4. Brose, K. and A. Lee (2008). "Cancer-associated thrombosis: prevention and treatment." Current Oncology 15(Suppl 1): S58.
5. Pretorius, E. (2018). "Erythrocyte deformability and eryptosis during inflammation, and impaired blood rheology." Clinical hemorheology and microcirculation (Preprint): 1-6.



Biography (150-word limit)

Dr Bester has been establishing her research group as well as her research focus since 2015. Her research focusses mainly focus on vascular complications, specifically hemorhology in chronic inflammatory diseases such as Type 2 Diabetes, breast cancer and prostate cancer patients. She uses novel techniques that distinguishes her from most of the research in her field. Her goal with her research is to improve tissue perfusion to improve wound healing as well as quality of life in patients with vascular complications as well as to determine the thrombotic risk in a specific patient population.

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Notes/Comments: