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Case Report

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# Prolonged Thrombin Time in Asymptomatic Patient with Hypo Dysfibrogememia Tucson

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## Abstract

A 61 years female patient with known diagnosis of the breast cancer in remission for more than 10 years has Renaud's disease. During her work up for lupus and lupus anticoagulant which both were negative a prolonged thrombin time was noted which was done by mistake. She has no history of bleeding or thrombosis and last recent surgery was 5 years ago for spinal stenosis and was uncomplicated. Her clinical examination is normal without evidence of any spontaneous bruises but colder hands. The thrombin time was greater than 125 seconds on two different occasions and correction of it by addition of normal plasma was down to 56 seconds and was thus incomplete. Her prothrombin time and PTT were normal and there was no evidence of FDP or D-Dimers. There was no evidence of circulating heparins. The fibrinogen level was normal. The para proteinmia was excluded by normal serum protein electrophoresis and by immunofixation . Thus it is felt that this patient has dysfibrinogenemia or hypo dysfibrinogenemia without bleeding or thrombotic complication. The literature review shows approximately 55% of dysfibrinogenemia patients do not have bleeding or thrombotic complications.

## Case History

This patient with stage 1 B breast cancer treated with adjuvant chemotherapy more than 10 years ago developed Renaud's disease with progressive symptoms upon exposure to the cold. She has been followed by the rheumatologist and her work up lupus and the rheumatoid arthritis was negative. The work up for the lupus anticoagulant was negative and laboratory did additionally thrombin time and found it was abnormally high. By adding normal plasma to her plasma the thrombin time stayed abnormal but the correction was almost 50% from 132 seconds to 56 seconds. These tests were repeated twice with the similar findings. Her clinical examination has been normal without

presence of any ecchymosis or bruises and no evidence of any recurrence of the breast cancer and only colder hands.

## Methodology

The reptilase time was normal but there was no heparin or heparin like substances in the blood. The fibrinogen assay was normal. The FDP and D-Dimer were absent. There was no evidence of DIC as platelet count hemoglobin and white blood cells were normal. The review of the peripheral blood did not reveal the schistocytes or helmet cells. There was no defect in the fibrin polymerization by the paraproteinemia as serum protein electrophoresis with immune fixation was normal and there was no rheumatoid factor and her ANA levels were normal. The addition of the normal plasma did not correct the thrombin time on two different occasions.

## Discussion

Recently patient fell down in a department store sustaining large bruise on the forehead and on the face. She was seen in the ER and subdural hematoma was excluded though D dimer was slightly high. She had uneventful recovery from this. This case I believe is an example of hypo dysfibrinogenemia. In this disorder there is low level of dysfunctional fibrinogen where as in dysfibrinogenemia [1] there are normal levels of functionally abnormal plasma fibrinogen. Both these conditions are heterogeneous caused by many different mutations in the three fibrinogen encoding genes. Both are autosomal dominant disorders. The fibrinogen is a hexamer each gene is secreting two chains hence there are six chains. The fibrin network will have multiple copies of the molecule. The heterozygosity for one mutant allele is sufficient to impair the structure and function of the fibrin clot

In patients with dysfibrinogenemia large number of patients has phenotypic and genotypic [2] abnormalities. These disorders are labelled with the city where it was diagnosed. Majority of these patients have either thrombotic or bleeding complications.

In hypo dysfibrinogenemia like our patients there is low level of dysfunctional fibrinogen. The one mechanism is the heterozygosity of single mutation that leads to synthesis of an abnormal fibrinogen chain which is secreted less efficiently than the normal fibrinogen like fibrinogen Koyoto.[3] As our patient has normal fibrinogen she probably has heterozygous mutation. In homozygous mutation like in fibrinogen Otago or Marburg [4,5] there is reduced secretion of functionally normal fibrinogen. I hence label my patient as first case of Fibrinogen Tucson, with hypo dysfibrinogenemia.

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