The metabolic syndrome is a common, very complex set of heterogeneous risk factors that are interlinked and increases the prevalence of atherosclerosis and type II diabetes mellitus and chronic inflammation. The subsequent atherosclerosis and consequent inflammation in turn increases the risk for thrombo-embolic stroke and myocardial infarction. Both thrombo-embolic stroke and MI are caused by the formation of pathological thrombi or where an atherosclerotic plaque is ruptured resulting in an occlusion of a blood vessel. Transient ischemic attack (TIA) in contrast to thromboembolic stroke is caused by the temporary occlusion of a cerebral artery resulting in neurological symptoms typically lasting less than one hour. Several studies have been conducted on the relation of metabolic syndrome and TIA, but very few have focused on the alterations resulting in TIA. In this study the morphology of the formed thrombus is examined to determine what changes take place in the coagulation characteristics of metabolic syndrome affected individuals and how this alters the structure of the clot.

Participants in this study included a control and experimental (TIA) group. After written consent was obtained 5 ml of blood was drawn. Venipuncture of TIA patients was done within 48 hours after symptoms developing. Whole blood and platelet rich plasma (with and without thrombin) was prepared following standard preparation protocols for SEM and AFM. SEM was used to investigate red blood cell (RBC) and platelet morphology, platelet activation and fibrin fiber arrangement. AFM was employed to examine RBC morphology, roughness and mechanical properties. Furthermore platelet and fibrin fiber mechanical properties were also studied.

Preliminary results indicate increased platelet activation and aggregation with decreased elastic modulus. The fibrin networks from TIA patients appear more disorganized and curved when compared to controls. No RBC shape changes can be seen when using SEM, however, when whole blood was artificially activated by the addition of thrombin, the RBCs of the control group show dramatic alterations in shape due to the presence of fibrin fibers exerting a force on the RBCs. These findings are corroborated by AFM results indicating increased Young's modulus and significant changes in the 1st and 3rd order membrane roughness indicating changes in the membrane macromolecular composition.

Fig. 1: RBCs of experimental group. A: SEM, scale 1µm. B: AFM, scale 1µm. C: Surface Roughness Image, scale 200nm. D: Surface profile original and 1st-3rd order.

Fig. 2: Platelet morphology. A: SEM, scale 1µm. B: AFM, scale 1µm.

Fig. 3: Fibrin network arrangement and morphology. A: SEM, scale 1µm. B: AFM, scale 1µm.

Fig. 4: Whole blood clot morphology in vitro, scale 1µm.