Editorial

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Unsolved Favorable Effect of Statin on Blood Viscosity

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In an interesting study, Jung et al.[1] found an effective reduction of whole blood viscosity in statin naïve acute coronary syndrome (ACS) patients with early statin administration. In statin naïve ACS patients they found an apparent decrease in whole blood viscosities at both of the two shear rates. The hypothesis that a lipid-lowering reagent, statin, contributes to blood viscosity decrease is very attractive and gave rise to immediate therapeutic possibilities. Jung et al.[1] claimed that this was the first report on the potential effect of statin on blood viscosity reduction. Using a capillary scanning method,[2] they compared diastolic blood viscosity (at a shear rate of 1 s^{-1}) and systolic blood viscosity (at a shear rate of 300 s^{-1}).

Generally, blood viscosity at low shear rates is strongly affected by red blood cell (RBC) aggregation, whereas blood viscosity at high shear rates is influenced by RBC deformability and plasma viscosity. It is commonly accepted that whole blood viscosity is an overall indicator reflecting RBC aggregation, RBC deformability and plasma viscosity. If there is any change among three components, the whole blood viscosity would be altered. Thus, one can expect that there would be apparent alteration of above hemorheological parameters. Unfortunately, plasma viscosity was not measured in the present study, without plasma viscosity information, it is difficult to determine whether the reduction of blood viscosity was caused by a plasmatic factor or cellular factor.

With careful reviewing the measured clinical data listed in Table 2,[1] both aggregation index (AI) and half time ($t_{1/2}$) showed no difference in Group II. AI represents the quantity of RBC aggregation, whereas half time does aggregation kinetics. In general, the high value of AI accompanies the small value of half time. Also, fibrinogen, which is the strongest factor in RBC aggregation, was not significantly changed between baseline and one month for Group II. For deformability, there was also no significant difference of EImax. However, half shear stress ($SS_{1/2}$) was increased in Group II (from 2.08±0.14 to 2.25±0.28). The increase of half shear stress indicates a decrease of RBC deformability.[2] This means that RBC deformability was decreased with statin administration. However, systolic blood viscosity was decreased with statin administration. Therefore, an explanation of the mismatch between viscosity change and RBC deformabilty change is unclear, and there was no hemorheological data to explain the reduction of blood viscosities at the two shear rates.

In Table 2,[1] white blood cell (WBC) count shows a significant correlation in Group II. In fact, there have been reports on a correlation between WBC count and whole blood viscosity.[3-5] Steinberg and Charm[3] examined blood viscosity with varying concentrations of WBCs. Furthermore, a WBC count was correlated with hypertension.[6] Therefore, it is required to examine the role of statin on a WBC count in future study.

In the study of Jung et al.,[1] platelet count was provided but the degree of activation and deactivation were not known. Upon activation by agonists or shear stress in flow, platelets were released in excess of 300 active molecules from intracellular granules.[7] These factors caused an increase in either the RBC aggregation[8] or whole blood viscosity. Therefore, it is also required to investigate the correlation between blood viscosity and platelet-released molecules.

Even though the results of Jung et al.[1] could not easily be delineated with the provided clinical and hemorheological data, its potential is high and further investigation is strongly recommended.

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References

1. Jung LY, Lee SR, Jung JM, et al. Rosuvastatin reduces blood viscosity in patients with acute coronary syndrome. *Korean Circ J* 2016;46:147-53.
2. Shin S, Ku Y, Park MS, Suh JS. Slit-flow ektacytometry: laser diffraction in a slit rheometer. *Cytometry B Clin Cytom* 2005;65:6-13.
3. Steinberg MH, Charm SE. Effect of high concentrations of leukocytes on whole blood viscosity. *Blood* 1971;38:299-301.
4. Sharma K, Puniyani RR, Bhat SV, Advani SH, Hegde U, Rao S. Blood viscosity parameter correlation with types of leukemia. *Physiol Chem Phys Med NMR* 1992;24:159-64.
5. Ho CH. White blood cell and platelet counts could affect whole blood viscosity. *J Chin Med Assoc* 2004;67:394-7.
6. Karthikeyan VJ, Lip GY. White blood cell count and hypertension. *J Hum Hypertens* 2006;20:310-2.
7. Golebiewska EM, Poole AW. Secrets of platelet exocytosis - what do we really know about platelet secretion mechanisms? *Br J Haematol* 2013 Nov 30. doi: 10.1111/bjh.12682. [Epub ahead of print]
8. Kim JH, Lee H, Lee BK, Shin S. Influence of shear stress on erythrocyte aggregation. *Clin Hemorheol Microcirc* 2015 Sep 25. [Epub ahead of print]