Plenary lecture (ESCHM) Tuesday, July 3,

09:00 - 10:00 room MLH-A+B

L1 Blood rheology: from exercise responses to sickle cell disease pathophysiology

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It will be a 3 parts talk:
1) Exercise and blood rheology:
Blood viscosity increases during exercise. This increase would be the consequences of the rise in hematocrit, plasma viscosity and red blood cell (RBC) aggregation, and the decrease of RBC deformability. The decrease of RBC deformability has been attributed to lactic acidosis and oxidative stress. However, we and others reported that RBC deformability can also increase during exercise in highly trained individuals, and this increase would be the consequence of a greater production of nitric oxide (NO) into the RBC.
2) Sickle cell trait (SCT):
SCT is the heterozygous form of sickle cell disease (SCD) and is usually considered to be a benign condition. However, large epidemiological studies demonstrated a higher risk for SCT individuals to collapse during exercise. At rest, blood viscosity and arterial rigidity are higher in SCT compared to control individuals. During exercise, blood viscosity of SCT carriers reaches very high values but adequate hydration has been demonstrated to offset this increase.
3) SCD:
SCD patients have abnormal hemoglobin (HbS), which polymerizes under de-oxygenation and causes the sickling of RBC. Sickle RBC are fragile and poorly deformable. Patients with the lowest RBC deformability are at higher risk to develop leg ulcers, glomerulopathy and priapism while those with the highest deformability have frequent vaso-occlusive crises (VOC). Any rise in blood viscosity increases the risk for VOC because vascular reactivity is blunted in SCD. Hemolysis, increased oxidative stress and the high amount of circulating microvesicles are involved in the development of vasculopathy in SCD. Enhanced eryptosis caused by oxidative stress would be the cause of RBC-microparticles genesis in SCD.