Atherosclerosis is a result product of the interrelation between blood and arterial vessel. Various kinds of systemic risk factors affecting the arterial wall are intermingled in complex cascades of interactions, such as environmental, genetic and biologic risk factors that cause atherogenesis and progression to atherosclerosis. Although many systemic risk factors predispose to its development, atherosclerosis preferentially affected certain regions of the circulation. This suggests that the lesion-prone areas may be, at least in part, due to biomechanical-related factors.

Furthermore, intraluminal hemodynamics, such as flow velocity, pressure changes, and wall shear stress (WSS) have been suggested to be additional risk factors for the development of coronary atherosclerosis. WSS is a the product between blood viscosity and the shear rate. According as the classic concept of physics, volume flowrate in tube structure is affected mainly by pressure difference and resistance, and the resistance depends linearly upon the viscosity and length, but the fourth power dependence upon the radius is dramatically different. So, I have studied the influence of WSS and blood fluiddynamics on the atherosclerosis using computational modelling and simulation, and the effect of blood rheology on the patients with ischemic heart disease using the measurement of whole blood viscosity, plasma viscosity, and RBC aggregability.

1. Flow velocity and WSS profiles at the coronary artery (Fig. 1);

The velocity profiles of the diastolic acceleration and deceleration phases were represented as a pulsatile waveform of coronary circulation. Compared to the acceleration phase in which there was little flow reversal on the curved area of the left
coronary artery (Fig. 1a). The deceleration phase of coronary circulation demonstrated that the recirculation zone in the curved area was accentuated (Fig. 1b).

![Velocity profiles(cm/s)](image)

(a) acceleration phase  (b) deceleration phase

The distributions of WSS of coronary circulation were different between those in the acceleration phase and those in the deceleration phase. Geometrical analysis of WSS during deceleration phase showed that the highest value at the inner WSS, the lowest value at the outer wall. The most highly separated value of WSS in the anterior descending artery was more prominent during the deceleration phase.

2. Flow velocity and WSS profiles at the abdominal aorta (Fig. 2);

Compared with the results in coronary artery data, skewed changes of velocity were noted. It makes a flow reversal at the outer wall of the bifurcation branch during the deceleration phase. The separated and reversed wall shear stress at the outer wall around the branched site was noted. The distributions of WSS during pulsatile flow were reversed between the inner and outer walls at the bifurcated branch according to the acceleration
and deceleration phase. However, the WSS during the cardiac cycle was not obviously different from that in the coronary model.

3. Blood Rheology in Stable Angina and Acute Coronary Syndrome: a Logical Basis for the Transition?

As demonstrated in Figure 3, RBC aggregation index \((M\text{ and }M_1)\) were elevated considerably in patients groups. It revealed 38.7% higher \(M\), 41.6% higher \(M_1\) values in patients with stable angina(SA), 62.9% higher \(M\), 71.1% higher \(M_1\) values in unstable angina(UA) compared to the controls \((p<0.001)\). ESR results of the patient population showed 128.8%, 215.8%, and 326.6% higher in order of SA, UA, and acute MI(AMI) patients group than control subjects \((p<0.001, \text{ Fig. 3a})\), and then it revealed much higher in acute coronary syndrome(ACS) comparing to SA \((p<0.002)\). It also indicated enhanced rouleaux formation and thus sedimentation in autologous plasma further confirming the Myrenne data. Furthermore, ESR can represent the severity of RBC aggregation in extraordinarily high aggregator of AMI patients. As anticipated, all samples exhibited non-Newtonian flow behavior with viscosity values decreasing as shear rate increases. Patients blood demonstrated significantly elevated viscosity values at almost all shear area,
when compared to the control samples in order of control < SA < UA < AMI (p<0.01, Fig 3b). Among the patients, ACS patients blood demonstrated significantly higher viscosity values at shear rates below 27.7 s⁻¹, comparing to SA patient (p<0.01, Fig. 3b).

Fig. 3. RBC aggregation indeces and whole blood viscosity in ischemic heart disease patients (SA: stable angina, UA: unstable angina, AMI; acute myocardial infarction)

4. Abnormal hemorrhological parameters in cardiac syndrome X (CSX) as a possible cause of microcirculatory dysfunction

CSX is characterized by several hemorrhological abnormalities known to adversely affect blood flow, in particular flow in the coronary microcirculation. Such abnormalities might complement other pathogenetic mechanisms, including higher mean TFC and hence sluggish coronary blood flow, thus leading to the development of microvascular ischemia and angina. The routine measurement of selected, easily assessable hemorrhological parameters may thus be of value for the clinical diagnosis and follow-up of this clinical entity. Note that the increased RBC aggregability for CSX patients (Fig. 4a) and whole blood viscosity (Fig. 4b) has important pathophysiological implications: given their greater sensitivity to pro-aggregatory molecules, changes of fibrinogen or other large protein concentrations in the plasma will have a bigger effect on RBC aggregation.
The present study showed that the rheological behavior of blood from various kinds of patients group was characterized by increased low shear blood viscosity, enhanced RBC aggregation, and was related to an altered intrinsic tendency of RBC to aggregate. Our findings suggest that abnormal hemorheological parameters may contribute importantly to the etiology of atherosclerosis and microvascular circulatory disorders. And we suggest that the rheologic parameters can be used for diagnosing and primary prevention of ischemic heart diseases. Further, they indicate the possibility of therapeutic measures aimed at normalizing blood rheology (e.g., plasma phaeresis) and hence microcirculatory flow.

Reference

