Arterial hypertension and stroke patients present higher erythrocyte adhesion forces, contributing to fibrinogen-dependent cardiovascular risk

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The increase of erythrocyte aggregation by high fibrinogen levels may be an indicator of cardiovascular risk. $\gamma'$ fibrinogen variant has been considered as a possible player in enhancing aggregation. Here, we assessed, at the single-cell level, the influence of fibrinogen on erythrocyte aggregation in essential arterial hypertension (EAH) and in stroke, as well as how it constitutes a cardiovascular risk factor in both diseases. We also aimed at understanding how $\gamma'$ fibrinogen is altered on these patients.

Differences on fibrinogen-erythrocyte interaction and cell-cell adhesion forces were evaluated by atomic force microscopy (AFM)-based force spectroscopy, with cells from 31 EAH patients, 20 stroke patients and 15 healthy blood donors. The main procedures used were previously described by us [1-4]. Results were correlated with patients’ clinical profiles.

AFM data show that the work and force necessary for erythrocyte-erythrocyte detachment is higher for patients than for healthy donors, with these parameters further increasing in both groups when higher fibrinogen concentrations are present. Fibrinogen-erythrocyte (un)binding forces were also higher in EAH and in stroke patients, when compared with the control group, despite a lower binding frequency [5]. The results can be associated with changes in blood flow, due to transient bridging of two erythrocytes by fibrinogen, representing an important cardiovascular risk factor. $\gamma'$ fibrinogen may contribute for the increased risk in both diseases, as we demonstrate that its levels are significantly increased in these patients’ blood. Our results may be relevant for potential future drug interventions to reduce erythrocyte aggregation and enhance microcirculatory flow conditions in cardiovascular patients.


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