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Short Note On Alpha-2-Macroglobulin

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Introduction

Exposure of human blood to artificial surfaces in clinical settings is usually related to an increased risk of thrombotic and inflammatory reactions [1],. These complications arise from a posh inter-play between surface induced protein adsorption, cell adhesion, and a series of blood protein cascades: the contact system, coagulation, and therefore the complement system [2], [3] Protein adsorption of contact factors to artificial devices or the action of tissue factor from blood cells has been suggested to be initiators of coagulation. Activation of the complement system induced by artificial surfaces is reportedly mediated through the choice and classical pathways and therefore the contact system [4]. Currently, the utilization of biomaterials in clinical settings require administration of anticoagulants to attenuate thrombotic complications. Such an approach, however, doesn't completely prevent the complications triggered by interaction of blood with artificial surfaces. Furthermore, studies investigating inhibition of the complement cascade or the contact activation system, as a replacement approach to enhance the blood compatibility of biomaterials, highlight the complexity of the interaction between blood and artificial surfaces .Investigations of the blood compatibility and development of latest biomaterials therefore involve the utilization of several assays measuring initiation of the contact system, coagulation, and therefore the complement cascade also as markers for platelet and leukocyte activation. The International Standardization Organization standard describes variety of assays to review specific analytes that reveal the activation status of those biological systems. However, no global and sensitive immunoassay exists for detection of activation of proteases induced by biomaterials. Such assay might be useful in search of biomaterials with improved blood compatibility. The broad-spectrum inhibitor, α2-macroglobulin (α2M), interacts with a huge number of proteinases in blood including those involved in touch activation and coagulation [9]. In human blood, α2M circulates as a 725 kDa tetramer during a concentration of about 2.4 mg/mL Upon interaction with a proteinase, α2M undergoes an irreversible conformational become a complexed or electrophoretic “fast form” (F-α2M) and exposes an otherwise hidden receptor-binding-domain (RBD) supported this, we hypothesized that detection of F-α2M might be sensitive and global approach for evaluation of the blood compatibility of biomaterials; a rise in F-α2M levels would indicate activation of proenzymes from one or more enzymatic cascades. we generated a F-α2M specific antibody and applied this in an enzyme-linked immunosorbent assay (ELISA) setup to live F-α2M in plasma.[5] Furthermore, we investigated whether the F-α2M ELISA could detect activation of calcium dependent and in-dependent proteases in plasma after incubation with artificial surfaces[6].

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