Damage of Granulocytes in Filtration Leukapheresis (FL)

To The Editor:

To elucidate the mechanism of granulocyte damage during FL, studies have been reported of the interaction between nylon-wool fibers and isolated granulocytes suspended in protein free media,1,2 in 5% plasma,3 or in 1% human serum albumin.4 In FL, however, heparinized whole blood is passed through a nylon-wool filter, resulting in plasma protein coating of the fibers within seconds.4 Cell adhesion to a plasma-protein-coated surface is a biologically active process that is fundamentally different from the passive and very firm attachment of cells to a naked, synthetic surface.5,6 Consequently, the interaction of nylon fibers and granulocytes in a protein-free medium is not a relevant model for granulocyte-fiber interaction during FL. In the presence of undiluted plasma, we observed no passive adhesion of granulocytes to glass, whereas 10% plasma and 1% human serum albumin reduced, but did not by far abolish passive adhesion.7 Wright and coworkers2 observed less lysozyme release by granulocytes attached to nylon fibers in the presence of 5% plasma than in a protein-free medium. This may be due to reduced, but probably not abolished, passive adhesion by plasma proteins coating the fibers.

Even experiments with isolated granulocytes and fibers, sufficiently coated with plasma proteins may be irrelevant to FL. We have demonstrated that granulocytes are retained from whole blood in glass-bead columns by a platelet-granulocyte interaction.8 Conceivably, this holds for nylon-wool fibers as well.

REFERENCES


To the Editor:

We believe this letter is important in one respect: It points out that clinically similar patients may have etiologically different processes and that potentially harmful treatment should only be initiated when there is strong evidence that the possible benefits outweigh the adverse effects. Unlike our patient, the patient described by Sieff and Chessells had evidence of abnormal or deficient erythroid-committed stem cells (no growth of CFU-E or BFU-E) and no evidence of an environmental defect (no serum inhibitor). Cytotoxic therapy in a patient with abnormal or deficient stem cells is extremely risky. We strongly concur with Sieff and Chessells that in children clinically similar to the one described in our article, an inhibitor of erythropoiesis should be demonstrated prior to cytotoxic therapy, but equally important in such children is the presence of a normal stem cell compartment. We would hope that publication of this letter would disuade others from the use of cytotoxic, immunosuppressive agents in clinically similar children without firm laboratory evidence of normal erythroid-committed stem cells and immunologic attack against these cells.

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