CORRESPONDENCE

Plasmapheresis in Thrombotic Thrombocytopenic Purpura (TTP)

To the Editor:

Use of plasmapheresis in the management of TTP by Bukowski et al. resulted in benefits to their patients felt to stem from removal of a toxic substance from the plasma.\(^1\) Repeated plasmaphereses were performed on the Aminco continuous-flow centrifuge. Two to three liters of plasma were removed and replaced with fresh-frozen plasma without reported unfavorable effects.

We have also attempted plasmapheresis of a 39-yr-old black male with TTP but were forced to suspend the procedure because of an acute hemolytic reaction. He had been admitted 5 days previously to another hospital with abdominal pain, fever, microscopic hematuria, and gastrointestinal bleeding. TTP was diagnosed on the basis of rapidly progressing anemia, microangiopathic changes in the smear, and thrombocytopenia. He was treated with prednisone, dipyridamole, and aspirin and then transferred to our hospital, where he was not symptomatic. His hematocrit was 28% and his reticulocyte count was 9.2%. Smears showed polychromasia and numerous schistocytes. The platelet count was 33 × 10^9/liter. Coagulation studies were normal. A direct Coombs test was negative. Urinalysis showed 1+ proteinuria and 10-15 red cells per high-powered field. Blood urea nitrogen and creatinine were normal. Serum lactic dehydrogenase was 1088 IU/liter. Serum bilirubin was 2.1 mg/dl and serum protein was 6.2 g/dl, with an albumin of 3.6 g/dl. Serum electrolytes, including calcium, were normal. Prednisone, dipyridamole, and aspirin were continued and infusions of Dextran 70 were started. It was elected to defer splenectomy or exchange transfusion, and instead we began a course of plasmaphereses.

One day after admission, the patient underwent plasmapheresis on the Haemonetics Model 30 discontinuous-flow centrifuge, at which time he was alert and cooperative. His blood pressure was 144/88 mm Hg and his pulse was 88. After exchange of 1700 ml plasma with saline and 5% normal serum albumin (calculated 35% exchange), his condition deteriorated. He became agitated, disorientated, and vomited 100 ml of bilious material. No focal neurologic signs developed. The procedure was terminated. His blood pressure was then 130/80 mm Hg and his pulse was 100, and he had a serum sodium of 140 meq/liter, a potassium of 3.5 meq/liter, a CO2 of 25 mM/liter, and a calcium of 7.6 mg/dl. His hematocrit was 24%. Plasma in the collection bag had a brownish tinge and a hemoglobin concentration of 22.5 mg/dl.

One hour later, the patient developed gross hemoglobinuria and hematuria. Six hours later, he underwent an exchange transfusion, followed by a splenectomy. Urinary output remained good and hemoglobinuria cleared. Hemolysis continued, and 1 wk later he underwent a second exchange transfusion. Ten days after this second exchange transfusion, while he was maintained on a regimen of prednisone and dipyridamole, hemolysis stopped and the platelet count rose.

The role of the plasmapheresis in initiating this hemolytic episode is unclear. Differences between Bukowski’s procedure and ours are the type of cell separator and the fluid replacement. The Haemonetics Model 30 develops a higher centrifugal force than the Aminco and might damage TTP red cells. Increased red cell mechanical fragility is a feature of TTP.\(^2\) On the other hand, plasmapheresis for TTP using the Haemonetics Model 30 have been performed at the University of Connecticut\(^3\) and at Vanderbilt University with no evidence of hemolysis. Use of crystalloid and albumin replacement in plasmapheresis is established.\(^4,5\) and results of the blood chemistries show that the difference

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in replacement solutions also cannot explain either the hemolysis or the nonspecific neurologic symptoms observed.

Although this hemolytic episode during plasmapheresis may be coincidental, the dramatic change in this patient's condition cautions us. We encourage continued experimental use of plasmapheresis for TTP but feel that the effects of the various plasmapheresis procedures on these red cells should be studied.

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To the Editor:

The letter by Reiss et al. describing an acute hemolytic reaction in a patient with TTP undergoing plasmapheresis presents several interesting problems. The possibility that altered red blood cell mechanical fragility exists in TTP, and that this is an explanation for the observed reaction, certainly seems plausible and cannot be answered with any clarity at this point.

Our experience with plasmapheresis in a wide variety of other disorders has shown this procedure to be generally well tolerated and without significant side effects. We have employed this procedure with six patients with Goodpasture syndrome, two patients with aplastic anemia with blood group-incompatible donors in preparation for bone marrow transplantation, and four patients with Waldenström macroglobulinemia. We have not encountered any evidence of hemolysis in any instance; however, this certainly does not exclude this possibility in TTP. We would also agree that a procedure such as plasmapheresis should be considered investigational therapy in this disorder. However, in view of its apparent efficacy in the two patients reported, it would seem to deserve further attention as a therapy in TTP.

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R Reiss, V Shah, R Kalter and A Panlilio