REFERENCES

3. Huebers H: Unpublished data

Splenomegaly After Intensive Treatment of Hemophilia

To the Editor:

The article by Levine et al.1 concerning abnormal liver chemistries and splenomegaly in intensively treated hemophiliacs left both observations unexplained. Although their literature search “revealed no reports of splenomegaly associated with hemophilia,” I would like to call attention to a 1966 report from our clinic on therapy of hemophilia with fraction I in which splenomegaly was observed and the cause explained.2 The fraction I used contained contaminating immunoglobulins with anti-A and anti-B isoagglutinins in sufficient titer to cause mild hemolytic anemia in type A or B recipients. Patients who developed hemolytic anemia had splenomegaly, and splenomegaly subsided as hemolysis regressed when fraction I therapy was discontinued. We could not relate hemolysis to the high fibrinogen levels present after fraction I. Fractions currently used for factor VIII therapy, such as cryoprecipitate or commercial concentrates, contain erythrocyte isoagglutinins that could account for the splenomegaly observed by Levine et al., for they found that the “incidence tended to increase as a function of intensity of therapy.” It would be interesting if the authors could correlate splenomegaly with A or B blood groups and elevated reticulocyte counts in their patients.

It should also be noted that in our article2 a hemophiliac was reported with massive splenomegaly, probably secondary to long-term, unusually high transfusion and factor VIII replacement therapy for chronic hemorrhage complicating blood cyst formation.

N. RAPHAEL SHULMAN, M.D.
Chief, Clinical Hematology Branch
NIAMDD
Bethesda, Md.

REFERENCES


Splenomegaly After Intensive Treatment of Hemophilia: Reply

To the Editor:

Dr. Shulman has correctly called attention to his previous report of splenomegaly in two patients with acute hemolytic anemia that was attributable to the transfusion of anti-A or anti-B isoagglutinins. Hemolysis, however, was unlikely to have been the etiology of the splenomegaly observed in our hemophilic patients. None of the 26 patients with palpable spleens was anemic, none had a reticulocyte count higher than 1.2%, none had an elevation of serum bilirubin, and 50% had type 0 erythrocytes.

Of interest is an apparent decline in the overall incidence of splenomegaly seen by us at this year’s recent Hemophilia Comprehensive Health Clinic sessions, in spite of an increase in the overall intensity of infusions of plasma products. Whether the splenomegaly we have observed is due to the infusion of foreign, denatured, or aggregated proteins, is a function of the formation of antigen antibody complexes, is attributable to the emergence of latent cytomegalovirus, or represents any of the many other causes of splenic enlargement remains to be determined.

PETER H. LEVINE, M.D.
Director, New England Area Comprehensive Hemophilia Center
Professor of Medicine
University of Massachusetts Medical School
Memorial Hospital
Splenomegaly after intensive treatment of hemophilia [letter]

NR Shulman